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DISEASES OF THE KIDNEYS AND URINARY ORGANS

VOL. I.

TEXT-BOOK

OF

DISEASES OF THE KIDNEYS

AND URINARY ORGANS

BY

PROFESSOR DR. PAUL FÜRBRINGER

DIRECTOR OF THE FRIEDRICHSHAIN HOSPITAL, BERLIN; ROYAL MEDICAL COUNCILLOR
AND MEMBER OF THE ROYAL MEDICAL COUNCIL OF THE PROVINCE OF BRANDENBURG;
MEMBER OF THE IMPERIAL LEOPOLD-CAROLINE ACADEMY ("LEOPOLDINA");
MEMBER OF THE "CONGRESS FÜR INNERE MEDICIN," ETC., ETC.

ROYAL COLLEGE OF PHYSICIANS EDINR. LIBRARY.

TRANSLATED FROM THE GERMAN WITH ANNOTATIONS

BY

W. H. GILBERT, M.D.

PHYSICIAN IN BADEN-BADEN; PHYSICIAN TO THE SANATORIUM DDR. FREY-GILBERT,
BADEN-BADEN; MEMBER OF THE "CONGRESS FÜR INNERE MEDICIN";
MEMBER OF THE "BALNEOLOGISCHE GESELLSCHAFT," ETC., ETC.

WITH COMMENDATORY LETTER FROM

SIR THOMAS GRAINGER STEWART, M.D., F.R.S.E.

PHYSICIAN IN ORDINARY TO H.M. THE QUEEN FOR SCOTLAND; PROFESSOR OF PRACTICE
OF PHYSIC AND OF CLINICAL MEDICINE IN THE UNIVERSITY OF EDINBURGH;
AUTHOR OF "BRIGHT'S DISEASES OF THE KIDNEYS," "ALBUMINURIA,"
THE ARTICLES ON DISEASES OF THE KIDNEYS IN QUAIN'S
"DICTIONARY OF MEDICINE," ETC.

IN TWO VOLUMES
VOL. I.

LONDON
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TO

PROFESSOR DR. PAUL FÜRBRINGER

THIS TRANSLATION IS DEDICATED

AS A TOKEN OF ESTEEM AND ADMIRATION, AND IN

GRATEFUL REMEMBRANCE OF MANY ACTS OF KINDNESS,

BY

W. H. GILBERT.

University of Edinburgh,
January, 1895.

Dear Dr. Gilbert,

I cannot refuse to comply with your request that I should write a few words by way of recommendation of your translation of my friend Professor Fürbringer's work.

Professor Fürbringer has been known to the profession for many years as an able writer in various departments of medicine, and especially in connection with the kidneys and the urine. His special work upon the urine has attained in Germany a high and well-merited reputation, and now that it is about to appear in English I welcome it as a valuable addition to the many works dealing with the subject in our own language.

It is not to be expected that all the views advocated should commend themselves to every one who has worked in the same field, and it would not be difficult to indicate some divergencies from the opinions advocated in this work. Still the book gives a solid account of the topics dealt with, characterised by laborious accuracy both in literary and practical research, and I have much pleasure in commending it to the notice of the profession, both in this country and in America.

I am, yours faithfully,

T. GRAINGER STEWART.

PREFACE.

Professor Sir T. Grainger Stewart having done me the honour of writing an introduction to my translation of Professor Fürbringer's text-book, one would rightly deem it an assumption on my part were I to enter into a detailed preface.

In submitting this translation to the English reader, I will therefore merely limit myself to a very few words.

So many works of eminent English authorities on diseases of the kidneys and urinary organs already existing, a translation of a work on the subject might seem superfluous were it not from the pen of one of the greatest German authorities, who has made renal and urinary diseases a subject of special study for years. Such, however, being the case, I think that Professor Fürbringer's work, especially as it contains somewhat divergent views and opinions from those usually accepted in England, must offer much of interest to the English profession, and prove itself acceptable to students and practitioners of medicine.

After due consideration, I thought it advisable to follow the example of the French translators, Dr. G. Caussade and Dr. H. Hartmann, in publishing my translation in two volumes, the first being devoted to the strictly medical, the second to the more surgical part.

Finally, I must express my most sincere thanks to Professor Fürbringer for the permission to translate his work, and also for kindly suggesting the addition of footnotes, which I hope may prove themselves of useful interest. To Professor Sir T. Grainger Stewart I desire to acknowledge my gratitude for his kind introduction. Thanks are also due to Mr. H. K. Lewis for the great care he has exercised in the production of the work.

W. H. GILBERT.

Baden-Baden, *January*, 1895.

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DISEASES OF THE KIDNEYS.

GENERAL REMARKS.

It has become an established custom in all accounts of urinary pathology and therapeutics to prefix to the special part, which treats of the various renal and urinary diseases, their diagnosis, prognosis and therapeutics, a general part treating of the nature and importance of certain prominent symptoms in the quality and quantity of urine and in the reacting effects of urinary and renal disturbances on the organism in general. To these symptoms belong the presence of albumin, blood or hæmoglobin and renal casts in the urine, as also of dropsy and uræmia.

I. ALBUMINURIA.

Without taking into consideration the so-called false albuminuria, *i.e.*, that condition in which the urine contains albumin only in consequence of a mixture of blood, pus, sperma, etc., we will now only discuss the genuine albuminuria.

According to the old clinical view the expression albuminuria is identical with the presence of coagulable albumin in the urine, be it produced by serum-albumin (sereoalbuminuria, serinuria), be it by serum-globulin (globulinuria, paraglobulinuria), or by both substances combined, whereby the presence of fibrinogen is irrelevant. As recent researches prove, this latter case seems the rule, as indeed it might *a priori* have been explained from the great facility of diffusion and filtration of the serum-globulin. *F. Hofmann* even suggests for the albuminous urine the idea of an albumin quotient varying from 1 to 10, a quotient the value of which is found by dividing the quantity of the globulin by that of the albumin. In some cases even the globulin forms the total of the albumin found in the urine. Now-a-days in testing for albumin we completely ignore the presence of globulin, as no importance for diagnosis and prognosis can be attached to it, and our methods for ascertaining its presence and quantity being so uncertain (*Hammarsten*, *Kamenski*, *Ott*, *Viglezio*). For testing whether urine contains albumin various procedures are employed:—

1. One warms a quantity of filtered urine in a test tube, adding 30 per cent. *nitric acid*. According to the quantity of albumin

contained we obtain a mere haze, a dense white cloud or thick coagulated masses. This test is not always reliable, especially not for the beginner, as in certain cases (rare ones it is true) the coagulation may be mistaken for such produced by resinous substances, *i.e.*, resinous acids resulting from the decomposition of aromatic salts (*Löbisch* and *Rokitansky*). Furthermore the dark colour of the urine often prevents our detecting slight hazes. In such cases *Roberts* advises to add a concentrated solution of sulphate of magnesia, which prevents the too strong decomposition of coloured matter. Nitric acid is also used as albumin test without heating the urine. About thirty drops of strong nitric acid are placed in the bottom of a test tube and then an equal quantity of urine is floated gently over the surface of this acid. At the line of junction a zone of coagulated albumin is developed. A fallacy may arise by the appearance of a zone of hydrated uric acid in highly acid urines or amorphous urates in neutral urines being developed at the line of junction. In these latter cases, however, the lower line of junction is indistinct and the upper one hardly developed. The fact of urates producing the typical zone can be avoided by diluting the urine with an equal quantity of water.*

2. Filtered urine is heated to boiling point, and to every 10 c.c. about five drops of diluted acetic acid are generally added. It is necessary to neutralise alkaline urine before filtration. This modification proves to be a very sensitive test and in no way discolours the urine. A still more sensitive test can be obtained if we add an equal volume of a concentrated solution of *Glauber's* salts (*Panum*) or one third part of a concentrated solution of chloride of sodium (*Heynsius*). Acetic acid must then be freely added and the whole heated. By this excellent method we avoid the danger that a surplus of acetic acid redissolves the albumin. Precipitation of mucin hardly ever occurs.

3. Albumin is precipitated by adding acetic acid and potassium ferrocyanide to the urine. Numerous comparative tests have proved that the following proportions are the best: To 10 c.c. of urine and eighteen drops of acetic acid add one to two drops of a ten per cent. solution of potassium ferrocyanide. This test, so sensitive for diluted urine, has proved inefficacious in a certain number of concentrated urines. A minute generally elapses before coagulation takes place.

Rapid processes.—The necessity of simplifying the tests of urine has led to the use of picric acid (*Bouchard*, *Galippe*, *Johnson*, *Esbach*, *Ralfe*), trichloric acetic acid (*Raabe*) and metaphosphoric acid (*Hindenlang*, *Wikstrand*). *Pavy*, who does not consider the ebullition and slow solution of the last-named acids as sufficiently reliable, has had globules and tablets made of citric acid and natrium ferrocyanide, which he pulverises and adds to the urine. *Tanret* recommends a mixture of corrosive mercury, potassium iodide and acetic acid. Among the various prepared test papers *Wilson* and *Geissler's* are the most popular. They are an invention which has done much to facilitate the testing of urine at the bedside. Two papers are necessary for this procedure ; one is impregnated with potassio-mercuric-iodide, the other with citric acid. Almost all these methods are simple and sensitive in general, but, as *Penzoldt* first proved, not reliable, as they sometimes lead one astray by producing coagulation in cases when the urine is not albuminous * (according to *Johnson* this coagulation is produced by mucin). *Fleischer* is much in favour of using corrosive mercury and succinic acid.

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* This fallacious zone also disappears when heat is applied. On addition of nitric acid to highly concentrated urines, crystals of urea nitrate may be formed; heat likewise dissolves them. Provided these precautions are taken I find this method to be one of the most reliable for practical use.—W. H. G.

When the urine can be analysed in a laboratory, hospital, or in the physician's consulting-room, the old tried tests are to be preferred. A different thing when the analysis has to take place without filter or lamp at the bedside of a patient, for instance, in the country, or when we have to execute a greater number of tests for purely diagnostic purposes. In such cases the much occupied physician must content himself with rapid methods as a preliminary. We should not hesitate a moment in recommending for such cases, prepared papers, if (quite apart from their quality of precipitating alkaloids) the fact of placing and extracting the papers twice in the urine did not in so far cause a disturbance, inasmuch as the soft paper leaves numerous fibres, and the extracting of these shreds soaked in the liquid is most objectionable. To avoid this we have together with *Stütz* prepared small gelatine capsules filled with a mixture of hydrargyrinatrium-chloride, chloride of sodium and citric acid. By simply cutting off the ends of the capsule, placing it into a test tube containing about 5 ccm. of urine, we have a neat and clean method. These capsules share with *Geissler's* reagent the peculiarity of precipitating uric acid in very concentrated urine, which sometimes causes a slight non-albuminous haze. These cases, however, are of no intrinsic value to the practitioner. We do not for a moment pretend that our capsules (obtainable from *Stütz's* pharmacy in *Jena*) are suitable for such tests where very small quantities of albumin have to be taken into consideration. Even a physician unprovided with the necessary utensils will be able to make a preliminary test with *Heynsius'* method, for one finds in the poorest cottage a spoon, a light, table-salt and vinegar.

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* Alkali and acid albumin peptones, alkaloids and urates are precipitated. Discrimination between these is however easy by application of the heat tests; if the precipitate consists of either peptones, alkaloids or urates it redissolves: when heated, on the other hand a fresh sample coagulating by heat shows it to be serum-albumin and not alkali or acid albumin.—W. H. G.

The question of distinguishing albumin and mucin precipitations, which *Reissner* discussed very fully twenty-nine years ago, has been recently again brought before us. The question itself is of no great practical importance on account of the minimal quantity of dissolved mucin contained in clear urine. The fact must be kept in mind, that even highly diluted urine betrays the presence of mucin through the plentiful addition of acetic acid, thus it is necessary in such a case to control the acetic acid heat test with nitric acid procedure. *Johnson* declares that picric acid only then precipitates mucin when another strong acid is present. Albumin very often accompanies dissolved mucin. *Senator* and *Citron* have latterly called one's attention to the fact that the products of decomposition of the cellular substance (nuclein) especially in alkaline and cystitic urines are precipitated by acetic acid, and like mucin remain insoluble in a surplus of acid. *F. Müller* has already shown the presence of a proteid substance precipitable by acetic acid. In spite of *v. Noorden's* statement that he has often found mucin in the urine of healthy persons, *Senator* and *Citron* conclude that only insignificant traces of mucin are to be found in non-decomposed urine.

Quantitative estimation of albumin.—The best procedure of making an exact quantitative analysis is to coagulate the albumin by means of heat; collect, dry, and weigh it. For the various details we refer to the text-books and manuals of the following authors—*Neubauer* and *Vogel*, *Hoppe-Seyler*, *Hofmann* and *Ultzmann*, *Löbisch*, *Zülzer*, *Huppert*, *Salkowski*, *Leube*, and *Thomas*. The practitioner can in a great many cases forego an exact chemical analysis and can replace it by approximate estimation, by simply comparing the degree of coagulation in the test tube with other tests of which the quantitative result is known. *Esbach's* albuminimetre is of good service, as first proved by *P. Guttmann* and *H. Schulz*. It consists of a graduated tube, in which the albumin is precipitated by a solution of picric and citric acid. The results, however, are less reliable the more albumin the urine contains, the more the specific gravity is increased and the lower thet emperature is (*Czapek*). *Zülzer's* "Uroscope" is a small tube in form of a U and is based on the similar principle; the albumin is precipitated by solution of concentrated chromic acid. Beginners usually overrate the quantity of precipitated coagulum; it may serve as necessary information that even a decided cloudiness, which as deposit totally fills the concavity of the test tube, is not above 0·1 per cent., that 1 per cent. albumin renders the urine completely opaque and produces a deposit filling up half of the tube.

If containing three and more per cent. the urine generally becomes coagulated *in toto* and the masses of albumin brown.*

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* Weighing is, as already mentioned, the only way of obtaining an exact quantitative estimate of albumin, for one must always remember that all

Certain French authors follow *Bouchard's* theory, distinguishing (nephritic) albumin and (dyscrasic) albumin; the former is characterised by a flocky deposit, the latter by an intransmutable regular coagulation.

With regard to practical clinical diagnosis one can for the present do without this distinction. We will only remark that the peptone, which was found by *Gerhardt* in febrile diseases as latent albumin, *i.e.*, as a precursory sign of albumin, and as elements of pus in pyuria, may be found in urine during the resorption of purulent effusion of pulmonary exudates, etc. (*Hofmeister*, *Maixner*, *v. Jaksch*); the peptone, which does not become coagulable through heat, is thus hemialbumose, an intermediary between peptone and albumin; it was first remarked by *Johnson* in cases of osteomalacia and has since then been pretty frequently found by *Senator*, etc., under the most varied circumstances; sometimes it appears as the forerunner of albuminuria (*Lassar*) or together with it ("mixed albuminuria," *Senator*). *Posner* agrees with *v. Noorden* in supposing that a relationship exists between hemialbumose and the presence of sperma in the urine. For directions to discover the presence of peptone and propeptone in urine see the various treatises on urine tests; no particular value can, however, be attached to it.

Clinical signification of albuminuria.—Those days are long over when the existence of albuminuria, even without engorgement, passed for a characteristic of a renal malady. On the contrary, our firm conviction that at least a tenth part of all healthy persons excrete at times albumin with their urine has brought us to consider the question, whether albuminuria is to be considered in every case as a symptom of illness, or whether it is reconcilable to what is commonly known under the name of "*health*".

Physiological albuminuria.—*Senator* has shown himself to be a firm and staunch upholder of the theory of physiological albuminuria.* Uniting *Ludwig's* and *Heidenhain's* theories on the filtration of urine, he considers it as a mixture of the transudation of the glomerular vessels, poor in albumin, and of the non-albuminous excretion of the urinary canaliculi. According to him the incessant variation of the pressure and rapidity of circulation in the vascular glomeruli, the variability of the excretion of the tubuli uriniferi, suffices to explain the presence or the absence

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methods based on judging the amount from the depth of the deposit at the bottom of a tube in relation to the quantity of urine have two fallacies: (1) an individual sample is of a higher or lower specific gravity than another; (2) urates or phosphates, together with mucus casts, granular debris, etc., collect together at the bottom of the tube and thus swell the bulk of deposited albumin.—W. H. G.

* Senator declares the quantity of this physiological albumin to be capable of reaching the quantity of 0·4 to 0·5 per litre.—W. H. G.

of physiological albuminuria, according to time and period when the test is made. This theory of *Senator* has found great support, through the certain proof of traces of albumin in the urine of a normal person (*Posner* and *Leube*), as well as through the results of *Adam's* experiments, who discovered albumin in healthy dogs between *Bowman's* capsule and the glomerulus, and finally by the latest experimental tests on dogs' kidneys made by *Munk* and *Senator*.

Nevertheless, certain authors protest most energetically against the existence of physiological albuminuria, *i.e.*, the fact that albumin is normally excreted by the glomeruli. *Greenfield*, *Coats*, *Middleton*, *Rosenstein*, *Grainger Stewart* and *J. v. Noorden*, conclude a number of systematic observations, with the opinion that in the most cases, in which one can prove albumin in urine of a patient, the careful further observation and examination of the urine gives decided proofs that certain, if only small, pathological processes take place in the uropoietic apparatus; *Johnson* is also of the same opinion as *Bartel*, that the presence of albumin in urine is in every case a pathological symptom. Even *Leube* has of late become much less liberal in accepting a physiological excretion of albumin with the urine. Whether, however, a physiological albuminuria in the above-mentioned sense exists or not, it must be considered as fortunate for the profession that complicated tests are necessary in order to prove albumin in normal urines, be it derived from the glomerular tuft, or from the intermixed formed elements. Were this not the case the diagnosis of albuminuria would soon lose every claim to being important, a fact which would under all circumstances produce most serious consequences. It is natural that the more sensitive the albumin tests are, the more cases there will be in which the physiological albuminuria (as simply quantitative exacerbation) can be proved. We, physicians, must therefore not endeavour to continually discover more sensitive re-agents, because, as we have seen, the reliability will then suffer. *On the contrary, practitioners should adopt as much as possible the old and reliable procedures of testing for albumin, should not trouble themselves about small unimportant hazes, but should only reckon with decided deposits, which even at first sight show their importance.* Each year of practical clinical experience impresses us more with the importance of this rule.

But even for the case that a physiological albuminuria in the word's strict meaning does not exist, that is to say, that every one cannot be considered to have albuminuric albumin, still there are at the present time such a number of positive experiences due to the fact of testing and observation on healthy individuals, suffering from more or less prolonged albuminuria, that we are in no way justified to consider the presence of albumin as a positive sign of renal disease. It is most important to remember that the presence of albumin in the urine is merely one particular symptom, and even in tolerable intensity proves no more kidney disease than, for example, a systolic murmur at the mitral valve proves organic heart complaint. The diagnosis depends on the quality and the clinical type offered by the various symptoms.

Classification.—Unprejudiced observation proves that it is impossible to strictly distinguish the so-called physiological albuminuria, and such which is produced by other non-renal diseases, from the excretion of albumin caused by coarse anatomical lesions. This difficulty is produced by the existence of various degrees and mixed forms of albuminuria. Nevertheless, we consider it necessary, in order to obtain a general view, to distinguish various forms, the classification of which should be as little schematical as possible. We distinguish, quite apart from the experimental view, five groups, of which we only describe two in this division :—

1. *Albuminuria* in *healthy* individuals, with and without special cause.
2. *Albuminuria* in different morbid disturbances where the kidneys are healthy, or at any rate free from evident anatomical lesions.
3. *Albuminuria* in febrile complaints.
4. *Albuminuria* with general and local hyperæmia ; albuminuria with stasis.
5. *Albuminuria* through *actual disease of the kidneys,* especially through the different forms of nephritis.

In opposition to the albuminuria known as transitory or accidental, one has placed the last form, called persistent albuminuria, as the former is often (1-4) of much longer duration than, for example, the albuminuria in acute nephritis. The distinction between nephrogenous and hæmatogenous albuminuria (*Stokvis* and *Bamberger*) is more successful, but the latter term is sometimes promiscuously used for the four first forms and sometimes only for excretion of albumin resulting from modifications in the sanguinary crasis.

1. *Albuminuria in healthy individuals with or without special cause.*

" As *Becquerel, Simon* and *Schmidt* have observed a number of *perfectly healthy individuals* exist who pass albumin in their urine at times, especially after heavy meals and violent exertion of the vascular system. I, myself, know two healthy young men who have the same phenomena." * So spoke *Frerichs,* nearly forty years ago, in his well-known monography on *Bright's* disease. The abundant experience of the last ten years proves this statement to be true. *Vogel* observed albumin in healthy persons for years (night-urine free). *Ultzmann* found albumin in a number of healthy, strong men, partly in consequence of bodily exertion and great excitement, partly so far periodical, that at times albumin was to be found at one time and an hour after it had disappeared; sometimes it remained for days and even weeks. *Leube* gives us the first precise idea of the relative frequency of the so-called physiological albuminuria through testing the urine of 119 healthy soldiers; five of these soldiers passed albumin directly on rising, and besides these, fourteen after a heavy march (or an average 0·1 per cent.). *Munn* discovered in a similar manner out of 200 healthy aspirants for life insurance twenty-four with albumin.* We ourselves have often found decided albumin in healthy, blooming young persons, with and without bodily exertion and without reference to meals having just been partaken of; further, in a home for children, out of sixty-one, seven with periodical albuminuria. Periodical albuminuria appeared at intervals of days and weeks, although during this time no change in the manner of living took place, and urine itself presented no variation in quantity and density. We watched the following interesting case for a considerable length of time: a young, apparently perfectly healthy physician, who had undergone violent mental excitement, felt pain in the kidneys; his urine (lessened in quantity) with increased specific gravity, contained 0·6 per cent. of albumin without remarkable morphological deposits. At intervals the urine was entirely free of albumin, and after about a year it returned to its normal condition. The connection of

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* *Herringham* describes in the *British Medical Journal,* 1891, in an article on " Cystical or Intermittent Albuminuria," a case of a boy thirteen years old, suffering from intermittent albuminuria. The only cause to which the albuminuria could be attributed was that the boy had remained too long in an *upright position.* Herringham explains this fact as blood pressure on the renal veins, which, together with weak circulation of the blood, causes venous engorgement in the kidneys. Connection of albuminuria with the effect of cold or

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muscular exertion was not to be found, nor had diet any effect. The greatest secretion of albumin took place in the morning before meat was eaten. Careful testing of the urine for acidity, urea and uric acid did not reveal any connection with the albuminuria.—W. H. G.

* How misleading physiological albuminuria can prove itself the following case demonstrates. A patient came under my treatment suffering from chronic diffused nephritis, the origin of which could be traced back to a severe cold caught some two years previously. In spite of all treatment patient succumbed to the disease a few months after leaving our watering-place. Shortly afterwards his widow communicated to me the fact that the assurance company her husband was insured in refused to pay the premium, the family physician incautiously reporting to have casually detected a trace of albumin six years before, although the company's physician had three years after this proclaimed the urine free of all abnormal constituents, and her husband, on the strength of this satisfactory examination, had been accepted. This, and various other facts, convinced me that the incriminated trace of albumin was merely of a physiological character, and I consequently advised the widow to take proceedings against the assurance company. This she did and the result was satisfactory, the court giving a verdict in her favour, as the company's own physician had not detected albumin and as the possibility of physiological albuminuria existed, and was thus in this case the most probable.—W. H. G.

psychological excitement with the secretion of albumin has been since this remarked by several authors, especially by *Johnson* and *Thomas.* Sometimes a really typical periodicity (precise quantities at precise times) is remarked, so that one speaks of an albuminous cyclus (*Pavy*, *Klemperer*, *Kinnier*, *Teissier*, *Merley*), but to designate such forms as characteristic clinical types, or even as constitutional illnesses of gouty origin, seems to us, in the presence of the numerous transitory forms which we have seen in the course of years, to be going rather too far. We are, however, somewhat inclined to doubt in *Pavy's* illness, temporary augmentation of the elimination of pigmentary matters of the urine, of albumin, of uric acid and urea.

Similar observations have been made by *Senator*, *Lépine*, *Leube*, *Kleudgen*, *Leroux*, *Engel*, *Mahomed*, *Grainger Stewart*, *Bull*, and others. Lately *v. Noorden* has brought us large and very remarkable statistics of his own observations; out of 53 soldiers and 112 hospital patients about a third had albumin. The fluctuations with regard to time and quantity were enormous. A part of the subjects of his first group (in which representants of our second category form the chief part) and possibly the majority of those who had his so-called *false* albuminuria (mucin and albumin in recrutes, produced by a slight catarrh of the urinary organs) may belong to the category of albuminuria in healthy individuals, while, in cases where deposits of renal epithelium and red blood corpuscles have been found (*Griswold* and others), he admits the existence of temporary circumscript processes in the kidneys. Well founded as his calculations for a regular maximum of secretion of albumin in the morning hours appear, all the more astonishing appears his denial of the effect of muscular exertion on albuminuria. This influence has been proved for a great length of time by a series of such indisputable facts and by such numerous observations that we have not for a long space of time observed albuminuria in healthy persons. These facts speak decidedly against *Noorden's* theory.

Grainger Stewart describes various benign forms of albuminuria; out of 407 healthy individuals a third had albumin, and he considers a heavy breakfast as one of the principal causes. We may reasonably add to this group cases of puerperal albuminuria (*Ingerslev*); albuminuria produced by wind instruments (*Grainger Stewart*, *Sterling*), as well as those cases following the use of cold baths (*Johnson*, *Chateaubourg*), and violent perspiration (*v. Rokitansky* and *Löbisch*). Finally we must not forget to mention the fact, that according to the experiences of *Virchow*, *Dohrn*, *Ruge*, *Martin* and *Ribbert*, a considerable number of infants suffer during the first days of their life from albuminuria. This evidence proves that a certain number of individuals, children as well as adults, can show, without signs of local or general disturbances, specially without any suspicion of disease of the kidneys, for a short time, or even for years, light intermittent, cyclic remittent or permanent albuminuria (generally under 0·1 per cent.); the etiology is in these cases impossible to be ascertained. A *morning maximum* is here often to be met with, but certain facts are nevertheless contrary to this rule, such as violent *muscular exertion*, violent *mental excitement*, heavy meals, cold baths and transpiration. All these incidents cause secretion of albumin, or, at any rate, influence it.* A description of the etiology of these cases will be found later on. Still we may here add that most of the already named causes generally increase secretion of albumin in genuine cases of renal disease, and thus undoubtedly have a pernicious effect on the kidneys. Sudden changes in the composition of urine do not appear in decided cases of organic kidney disease. A moderate quantity of hyaline casts is often found in healthy individuals and is generally of no importance. According to our experience, these formations are not always missing in the cyclic forms as is sometimes maintained. *Leube* warns against errors caused by the concretion of uratic salts in the form of renal casts. This author is of the opinion that when renal casts and albumin are present, a serious circulatory disturbance or irritation of the kidneys exists. He furthermore declares that, on entering more closely into the subject, we generally find headache, dyspepsia and other slight uræmic symptoms, which decidedly point to the existence of latent renal disease. On the contrary, in such cases the absence of every morbid symptom, entire disappearance of albumin and excellent health are ordinary appearances. Nevertheless, *Leube's* advice to exercise great caution as to forming a diagnosis is of great importance. The nephritic forms of albuminuria are the most frequent; secretion of albumin is always serious and deserves strict attention.

2. *Albuminuria in various derangements, but where the kidneys are in a healthy condition, free from anatomical lesions.*

To this class belongs principally excretion of albumin in *weak persons*, *nervous persons* and *anæmic persons*, especially in young persons. One often hears complaints about general depression, nervousness, headache, dyspepsia, also sometimes about diarrhœa;

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* A significant case of physiological albuminuria has been witnessed by myself. For years a patient excreted albumin after heavy meals and bodily and mental exertion. Most accurate tests proved the urine free of albumin at all other times. Patient came under my treatment and I could only corroborate these facts. The quantity of albumin at the before-mentioned times was between 0·3 and 0·5 per litre, while the urine was always free of morphotic elements. The typical periodicity, as well as the *general* healthy condition of the patient, spoke against nephritis, and I could accordingly console the patient, who was under the impression of suffering from a serious organic disease. One day albumin was no more to be found at the wonted times, and regular tests for weeks proved its total absence. Two years have now elapsed and albumin has no more been detected; having interested the patient's physician for the case I have received authentic reports.—W. H. G.

but even the most careful examination cannot prove organic disease. Although tests show traces of albumin, still indications are wanting of sufficient urinary disturbance to prove real disease of the kidneys (to be excepted are: cases where together with hyaline casts, epithelial casts, renal epithelium and even blood corpuscles are to be found). The course of the often chronic, but always curable, albuminuria resembles the cases described in the first group. This form of albuminuria has often a chronic character, but is always curable and generally corresponds with the group first mentioned (*Dukes*, *Moxon*, *Gull*, *Rooke*, *Edlefsen*, *Wagner*, *Fürbringer*, *Kinnicut*, *Rosenbach*, *Granville*, *Steven*, *Henderson*, *v. Noorden*). Other groups (*Johnson*, *de Mussy*, *Saundby*) belong partly to the protracted convalescence of scarlet fever nephritis, and latent renal atrophy. In one of the cases mentioned by *Falkenheim*, albuminuria showed itself chiefly when the patient was in a recumbent position; then a large tumour of the spleen compressed the renal vessels. In what degree such patients are to be considered as belonging to this group, or as suffering from nephritis, is not easily to be ascertained. They are generally recorded in the hospital journals (*Wagner*, *Fürbringer*) as patients suffering from nephritis, and their urine proves itself soon free from albumin in consequence of repose and appropriate nourishment. Albuminuria in weak and anæmic persons is more or less closely connected with the albuminuria, which is frequently observed in constitutional maladies (progressive anæmia, certain forms of tuberculosis, leucæmia, etc.), in apyretic chronic affections, especially when they are accompanied with serious anæmia, and with such caused by inanition and marasmus, or simultaneous with hæmorrhage (*Quincke*) with the terminal form observed some days before death (*Runeberg*), certainly also with such found during collapse. The same relation exists between (diarrhœtic) albuminuria (*Fischl*, *Stiller*), which we have observed several times in the course of apyretic intestinal catarrhs, without collapse. A part of these forms presents (as shown by the urine) the transitory forms between nephritis and cholera kidney. The type of albuminuria frequently found together with *diabetes mellitus* (according to *Pollatschek* 37 per cent.) can only be considered as belonging partly to this group. Its classification as dyscrasia (according to *Ebstein's* newest theory, the albumin diffuses itself in the urine, even if the globulin is free from carbonic acid) is in opposition to *Stokvis*' theory, who strongly advocates its dependence on local renal lesions. Secondly, the serious primary and secondary nervous diseases, with anatomical lesions of the kidneys existing, can produce albumin. We often met with it during epileptic convulsions (*Brünninghausen*), although *Huppert* goes too far in declaring that every attack produces transudation of albumin into the urine. On the contrary, according to the latest experiments of *Mabille*, *Saundby* and *Kleudgen*, who point out that mixture of sperm in the urine is taken for albumin, albuminuria is seldom a direct sequel to epileptic convulsions. The same appears to be the case in tetanus (*Kussmaul*) (tests in many of the cases have shown a negative result), while in cases of delirium potatorum, according to the observations made by *Fürstner*, *Weinberg* and *Näcke*, an average of 50 per cent. of albuminuria is to be found. Finally, mental diseases (*Köppen's* "central albuminuria"), concussion of the brain and shock, coma, painful paroxysmal affections of the abdominal organs (*Fischl*), such as cardialgia, violent colic, with and without collapse, may be considered as belonging to this category, as also albuminuria caused by saturnine colic (*Ollivier*). *Englisch* and *Frank* have recently given us interesting explanations on the appearance of albumin in cases of incarcerated hernia. Albumin was found in more than half the cases observed; they are of certain diagnostic value. Through compression of the thorax *Schreiber* has experimentally produced a form of albuminuria which may be considered as belonging to the engorged kidney.

So much for the most important clinical facts, later on we will treat of albuminuria in febrile maladies and engorged kidney. It is not necessary to impress its diagnostical and prognostical importance. We are, unfortunately, in spite of considerable experience, as yet unable to give a satisfactory pathogenesis. The scientific and learned discussion held some years ago in the Medical Association in Glasgow on this subject by celebrated authorities, did little service in the matter. The latest experiences have, however, demonstrated that it is impossible (as often endeavoured to show) to prove one sole basis for the origin of functional albuminuria. Granted that numerous forms of albuminuria exist, equally large is the number of deviating theories. For the present we will abstain from going into explanatory details on the already mentioned categories and limit ourselves to a short statement of the most important prevalent theories on albuminuria, with short reference to the experiments made, inasmuch as they are capable of explaining their etiology.

Various theories of albuminuria.—In order to explain the genesis of albuminuria one has had recourse to changes of renal circulation and to altered condition of the membranes of filtration * and of the blood itself, and thus to physical, biological and chemical derangement. With regard to the changes in renal circulation, nothing is more natural (after having accepted the general view on the theory of filtration and its pressure) than to make an increase of the arterial blood-pressure responsible for the transudation of albumin out of the blood into the urine.

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* Before accepting the view as a full explanation that the stratum of epithelium covering the glomeruli and lining the tubules retains albumin in health and prevents its transudation into the urine, further experimental inquiry is necessary. As *Hamilton* remarked, several membranes possess similar remarkable properties. The shell-membrane of an egg is permeable for a solution of sugar in one direction but not in the other, a grape skin has the same properties, during life the gall bladder retains bile and the aqueous humour is similarly retained by *Descemet's* membrane. Thus with regard to the passage of albumin, analogous protective influence may be claimed for the epithelial covering of the glomerular loops and lining of the tubules.—W. H. G.

Difficult as it is to establish clinical evidence that polyuria and hypertrophy of the left ventricle can alone produce albuminuria, still more is it the case to prove the influence of high pressure in the aorta. *Senator* argues correctly in declaring most of the experiments to be without intrinsic value. Thus albuminuria, produced by irritation of the cervical spinal cord, by strychnine, digitalis, and by other artificial methods, the object of which is to increase the blood-pressure in the aortic system, has no demonstrative value for the simultaneous spasm of the renal capillaries (*Grützner*), and impedes the conveyance of arterial blood. On the other hand, compression or ligation of the aorta, below the renal arteries, does not produce albuminuria (*Stokvis*, *Rosenstein*), even when blood-pressure is increased by simultaneous ligation of other large arteries (*Litten*). Neither section of the splanchnic nerve, nor that of the renal nerve, can produce transudation of albumin if one is careful to avoid secondary concomitant lesions (contusion of the vessels, etc., *M. Herrmann*, *Knoll*). *Lépine* made an interesting observation, after injecting salt-water into the venous system of a dog, transudation of the urine (without hæmoglobin) appeared; whether this fact is alone due to increased blood-pressure is uncertain. Our own numerous evidences prove that in cases of functional albuminuria this transudation accompanied by polyuria is absolutely diminished, yes, in some cases completely arrested by injection of salt-water; chemical tests confirm the disappearance of albumin. Latterly *Steven* and *Johnson* have come to our opinion and deny that the arterial pressure produced albuminuria. *Schmuziger* has tried in vain on himself to increase his albuminuria by introducing liquid into his circulatory system.

In direct opposition to the old dogma, that albuminuria depended on an increased pressure in the aorta, *Runeberg* made several experiments ten years ago on the filtration of albuminous solution through the animal membranes, and proved that the quantity of filtered albumin *increases* proportionally with *decrease* of pressure. In accordance with this theory we believe that in functional albuminuria the transudation of albumin, which is effected in the glomeruli, depends on a diminution of blood-pressure in the *rete mirabile*, *i.e.*, on an augmentation of pressure in the tubuli uriniferi. To proclaim that the various forms of albuminuria depend on the diminution of arterial pressure by applying the results obtained with inert intestinal membranes and also with pleura and ureter to living capillary walls, is a bold but fallacious attempt. In spite of the most ingenious inductions, *Runeberg* committed a serious error, not to mention the incongruity of his experiments with those made later on by *Gottwalt*, *v. Bamberger* and *v. Regéczy*). *Heidenhain* declares that the results attained by *Runeberg* could be differently interpreted; he proves that under the influence of filtration by increased pressure the transudation of albumin increases slower than the quantity of urine. Our own observations are opposed to *Runeberg's* theory in its exclusive sense. *Singer's* observation, that he found no albumin in the urine passed by a patient suffering from cholera nostras, shall have special attention later on. The urine was more plentiful doubtless before it became albuminous, due to the nutritive derangements of the glomeruli produced by the interruption of the circulation. We ourselves have seen at the conclusion of a very violent intestinal catarrh, which produced a retention of urine for twenty-four hours, 30 ccm. of excessively concentrated urine, without a trace of albumin. *Runeberg* nevertheless deserves the merit of having annulled by valuable experiments the old theory which attributes albuminuria to the increase of arterial pressure. We shall see that his theory holds good in a tolerably large number of cases of functional albuminuria, or rather, that is to say, it is in accordance with that one to which we give the preference. We mean the afflux of arterial blood to the glomeruli. Nearly twenty-five years ago *Stokvis* already affirmed from numerous personal experiences, that all circulatory derangements combined with obstruction of the arterial afflux or venous blood-current produce albumin. We know now, thanks to *Nussbaum's* experiments on frogs' kidneys, thanks to the microscopical examinations of albuminous kidneys after fixing the albumin at the point of transudation by boiling (*Posner*) or alcohol (*Ribbert*), that the glomeruli are (not without exception, *cf.* "Engorged Kidney") the principal centre for the transudation of albumin. The epithelial stratum of the rete pre-eminently opposes the transudation of albumin in normal conditions (*Heidenhain* and *Cohnheim*) in consequence of its excessive sensibility with regard to every retention of arterial blood in circulation; even the slightest derangements cause the loss of this faculty and render it pervious for albumin. Thus *Herrmann* and *Overbeck* observed albumin transude by a simple contraction of the renal arteries, and even through temporary interruption of the circulation, therefore the less arterial blood is supplied to the glomerular epithelium, the more albumin transudes through the vascular loops. This theory of *Heidenhain-Cohnheim*, with which *Posner*, *Litten*, *Charcot*, *Lépine* more or less agree, is based on a biological origin of albuminuria contrary to *Runeberg's* physical theory. It is apparent that in a number of cases diminution of pressure and retardation of the current in the glomeruli are coincident. This theory explains the majority of cases of functional albuminuria, combined with oliguria, in a very plausible manner, especially those forms in various anæmic affections (*Steven: "Deterioration of the Textural Structures, produced by Primary Illness"*), diarrhœa in the last stage of life, with decreased afflux of blood and albuminuria as sequel of serious nervous derangements. In most of the last-mentioned cases the vaso-motor derangements must also be considered as partly causal (*v. Bamberger*, *Capitan*), without, it is true, being always able to determine exactly the part which effects the irritation, reflex or direct, and that which depends on the paralysis of the renal vaso-motor or constrictor nerves (*Cohnheim* and *Roy*). We know that acupuncture of the fourth ventricle causes albuminuria by dilatation of the vessels and protraction of circulation (*Claude Bernard*, *Schiff*); coma, concussion of the brain, asthenia belong to the same category; they show, according to *Fischer's* theory of the shock (reflex paralysis of the splanchnic nerve with congestion of the large intestinal veins), transitional stage, which forms the link between them and engorged kidney, analogous to the painful paroxysms, which are so prevalent in abdominal affections combined with collapse and phenomena similar to the shock (*Fischl*). From the negative results of post-mortems *Bumm* attributes the albuminuria in delirium tremens to derangements of central innervation. *Johnson* has a peculiar theory for explaining albuminuria as a sequel to neurotic lesions. The spasms first affect the chylopoetic organs, and these produce secondarily, through excretion of the abnormal products of morbid circulation, the transudation of albumin, which therefore appears in a dyspeptic character. In a number of cases of epilepsy and tetanus, *respiratory derangements* and *muscular labour* are also associated with spasms of the *arteries of the kidneys*. The albuminous effect of the former, so generally and universally recognised, is still very difficult to explain. That it has nothing to do with an arterial reduction of pressure in the kidneys during the muscular labour, as *Runeberg* and *Edlefsen* assume, is certain, but the theory asserted by *Senator* of simultaneous diminution of water in the urine, is certainly not able to explain those cases in which bodily exertion considerably increases the quantity of albumin (according to *Schmuziger* as much as five-fold). One might conclude that transudation of albumin would be checked as a consequence of the augmented rapidity of circulation (*Oertel*). We should be inclined to attribute a lesion of the glomerular-epithelium in consequence of a kind of auto-intoxication through products of over-fatigue, if experience had not taught us that concussion of the body (as in riding and driving) more frequently plays a more important part than the real muscular labour. The attempt to make the changes of the vascular tonus (*Stewart*) responsible, as we believed ten years ago, only replaces in our opinion the former *non liquet* with a new one. *V. Rokitansky* and *Löbisch* have given us an interesting experimental illustration, that the transudation of albumin is dependent on intense perspiration. These authors proved the possibility of producing albuminuria through depletion of the renal vessels by means of pilocarpine, without disturbing circulatory statics. The changes, which in all these cases the glomeruli or rather their epithelium undergo, apart from the proofs of regenerative desquamatory processes of the glomerular epithelium in the fœtus and newly-born infants (*Rippert*), are unexplained. They are certainly generally very light and capable of quick compensation, for there is often, as we see, only an interval of a few hours between the passage of albuminous and non-albuminous urine. The cases in which albumin is transudated for a long time permanently without sanitary derangements or palpable cause are quite inexplicable. We are in these cases compelled to accept an individual permeability, be it of the said membranes (*Leube*), be it more with respect to altered conditions of the blood (*v. Noorden*). As already remarked, individual variations of the above theories appear unavoidable. One individual suffers from albumin immediately after exercise; another, in spite of the most violent epileptic convulsions, passes urine totally free from albumin.

In the third place *altered conditions of the blood* cause albuminuria. The important part, which these chemical derangements played formerly, before *Bright's* day, is now almost completely disputed. This fact is principally due to *Stokvis'* experiments; through them the original opinion that albuminuria is produced by hydræmia, hypalbumin and deficiency of blood and of chloride of sodium in the serum, can be regarded as extinct. Serum taken from nephritic individuals and injected under the skin of healthy animals was not able to render their urine albuminous. On the other hand, quantitative alterations in the composition of the blood through increase of salts, especially of chloride of sodium and urea with augmentation of the quantity of filtered albumin (*Kunkel*, *Senator*, *Hoppe-Seyler*, *Neumann*, *Lépine*, *Coignard*, *Mya* and *Vandoni*), are imputed to be the causes of albuminuria. The same is attributed to the introduction of fowls' albumin into the circulation, the albuminogenous effect of which *Stokvis* has proved in his experiments on rabbits, further to the incorporation of isolated albuminous matter of the blood into the circulation (*Estelle*, *Faveret*, *Lépine*). Thus the transudation of albumin produced by heavy meals (albuminuria alimentaria) meets a sufficient explanation. *Ebstein* considers the deficiency of carbonic acid as an etiological moment for albuminuria in cases of diabetes mellitus. According to his newest theory the fundamental cause of glycosuria is to be found in a deficient production of carbonic acid in the tissues which normally prevents the transformation of glycogen into glycose. *Senator* assumes the possibility of an extension of cerebral irritation, producing the same effect as acupuncture of the fourth ventricle. Nevertheless, as *Stokvis* observes, albuminuria in diabetes depends to a great extent on nephritic changes. It is certain that intense and lasting forms can exist, even after the disappearance of diabetes. *Rosenbach* declares that albuminuria appears as symptom of a surplus of free (non-combusted and non-combustible) albumin in the blood and as the sign of a "regulating" normal function of the kidneys. The *Semmola* theory of albuminuria and nephritis (fully discussed in the part on "Inflammation of the Kidneys") maintains that the albuminous substances in the blood have an increasing power of diffusion and a decreasing power of assimilation in consequence of derangement of the respiratory functions of the skin. Granted this view is correct, the urea contained in the blood or in the perspiration of individuals suffering from albuminuria would be lessened, a fact, however, which *Dockmann's* direct experiments in no way confirm. On the contrary, the greater number of results rather prejudice this somewhat arbitrary theory.

The latest experimental researches with regard to the albuminogenous effect of increased introduction of fowls' albumin have been almost always accompanied with negative result. Thus *Loewenmeyer* and *v. Noorden*, and formerly *Oertel*, were unable to prove this theory by injecting even great quantities of albumin. *Snyers*, to whom we are indebted for the latest experimental research on this subject, injected albumin and remarked a mere transudation of the injected substance, and by no means pathological albuminuria nor nephritis. *Hayem* came to the same result. Likewise *Tizzoni* and *Riva* discovered that the albumin contained in the urine is very easily assimilated, and that artificial albuminuria can only create temporary renal irritation.

We have sufficiently discussed albuminous dyscrasia, and its effects on serious albuminous disorders and organic kidney disease (see "Nephritis"). *Pavy* distinguishes between cyclic and renal albuminuria, he declares in cyclic albuminuria albumin is generally present, which can be precipitated by organic acids, while in the renal forms we are unable to precipitate the serum-albumin by these acids. The author's experiences in no way corroborate this characteristic fact. Finally *Grainger Stewart* and *Ralfe* have latterly drawn a parallel between the intermittent cyclic and similar functional forms of albuminuria, and between paroxysmal hæmoglobinuria. The last-named author, who has made great efforts at generalising, even considers these forms of albuminuria as abortive. Conditions which precede hæmoglobinuria are caused by an insufficient transformation of hæmoglobin in the liver to urea and pigment and then have a sequel of transudation of albumin. Hæmoglobin can be added to these three substances, when the insufficiency of hepatic labour reaches its height. The scarcity of the cases in which one can observe the transition of paroxysmal albuminuria to the corresponding form of hæmoglobinuria does not support *Ralfe's* theory.

Treatment.—A treatment for those forms of albuminuria, which do not endanger life, does not exist. Even *Saundby*, with his great experience, frankly owns to not having cured one single case by means of medicine; we have long ceased prescribing such. *Pavy* is of the same opinion. Hygienic measures, in the words' widest meaning, aid the spontaneous diminution of albumin, which, however, may last years. How albuminuria is relatively connected with fever, with engorged kidney and with genuine renal maladies is treated on in the special division of this book.

II. HÆMATURIA.

To prove admixture of blood to the urine (hæmorrhage or simple diapedesis), a very frequent and important symptom in the most varied maladies of the uropoetic system, we use the microscope (indispensable for distinguishing hæmaturia from hæmoglobinuria), spectroscopic examination, hæmin test and other chemical reactions. For different tests and the other characteristics of the hæmorrhagic urine, see special treatises on urine and analysis. *Levin* and *Posner* declare the use of the ordinary spectroscope (normal blood lines in fresh urine, spectre of methæmoglobin in hæmoglobinuria, signs of hæmatin in certain intoxication) to be erroneous, even common hæmorrhagic urines show signs of abnormal absorption. Quite pale urines can betray their contents of blood by the red colouring of the sediment. *Heller's* test suffices for the practitioner when there is only a suspicion of hæmaturia; one adds liquor potassæ to the urine and then boils it; the earthy phosphates precipitate the coloured matter of the red blood corpuscles and produce the reddish flakes which are only recognisable after some time standing; this test immediately follows that for albumin (acetic acid), when one generally remarks a dirty red colour of the coagulated masses of albumin. Precipitate of phosphates can be advantageously used for ascertaining hæmin. The presence of hæmaturia being proved, it remains to be decided whether the hæmorrhage proceeds from the *kidneys* or from the urinary passages. The distinction is often exceedingly difficult. If proceeding from the latter source (pelvis, ureters, bladder, urethra), blood coagula and characteristic casts are to be found. Hæmorrhage of the bladder often produces very hæmorrhagic urine; in syringing this organ the injected water reappears instantaneously coloured, while in hæmorrhage localised in the organs above the bladder this only occurs after several minutes. This procedure was first recommended by *Thompson* and later on by *Stein*, and has often disclosed the source of the hæmorrhage after all other differential signs have played us false. If a few drops of blood appear after emptying the bladder, it proves hæmorrhage to be localised in the neck of the bladder. Naturally, characteristic accessory symptoms (catarrh of the urinary passages, gall-stones, etc.) are of the greatest importance. The cystoscope often decides the question. Further details are to be found in the part treating on special pathology of the different organic diseases.

As a proof of the renal origin of hæmaturia we have several characteristic symptoms: uniform coloration of the urine at the beginning and end of micturition, blood casts, usually accompanied with renal casts, more or less advanced discoloration of the blood corpuscles produced by their longer retainment in the glomeruli and tubuli uriniferi, fine granular blood pigment in the sediment and increase of albumin. Finally renal hæmorrhage often produces in the course of the same day excessive variations in the hæmorrhagic character of the urine (*Guyon*). Besides this the symptomatology (dropsy, renal pain, tumour, etc.) of the primary maladies (trauma, nephritis, tuberculosis, cancer) characterises renal hæmorrhage.

For paroxysmal hæmaturia see the following part:—

III. HÆMOGLOBINURIA.

Although the presence of hæmoglobin as abnormal urinary constituent in disease of the kidneys is of inferior importance, we nevertheless make some short remarks on hæmoglobinuria, the clinical importance of which has only been lately proved. It offers diagnostical evidence for the existence of secondary renal disease. We will leave that form of hæmoglobinuria undiscussed, which accompanies hæmorrhagic nephritis.

The admixture of hæmoglobin with the urine was already considered two centuries ago as a result of transfusion of blood, but was, however, confounded with hæmaturia. Only a few years ago *Ponfick* recognised the real cause to be in the dissolution of the red blood corpuscles. We are now aware of the fact that hæmoglobinuria can appear under the most varied circumstances as an expression of hæmoglobinæmia. Apart from physiological disintegration of hæmoglobin the red blood corpuscles either crumble or the hæmoglobin is dissolved out of the stroma and enters the plasma in a dissolved condition. *Ponfick* showed later on that all forms of hæmoglobinæmia do not necessarily cause hæmoglobinuria, on the other hand it is dependent on a very active destruction of the red blood corpuscles. The spleen receives scoria of disintegration and becomes swollen ("spodogenous tumour"). Through the dissolution of the colouring matter of the blood, the liver and then the kidneys become tumefied; hypercholia manifests itself, and, after the absorption of one-sixtieth part of the total quantity of hæmoglobin, hæmoglobinuria results.

Etiology.—The special causes of hæmoglobinuria and specially those of hæmoglobinæmia are very numerous. To be mentioned are—

1. Real *poisons*, such as arseniuretted hydrogen (*Vogel*, *Wächtner*, *Eitner*, *Coester*), muriatic, and sulphuric acid (*Naunyn*, *v. Bamberger*), carbolic acid (*z. Nieden*), pyrogallic acid (*Neisser*, *Besnier*), pyrocatechin, hydrochinon, resorcin, naphthol, nitro-benzol, iodine, poisonous fungi (*Borström*), but especially *chlorate of potassium.* The hæmoglobinous effect of the last-named drug, formerly often confounded with nephritis diphtherica, was first discovered by *Jacobi* and experimentally and clinically proved by *Marchand.* Important practical significance must be ascribed to this discovery, inasmuch as relatively small doses can become extremely dangerous (about five grammes for children and ten grammes for adults). The poison has deadly effect by destroying the respiratory capability of the red blood corpuscles, through retention of urine in consequence of impregnation of the tubuli uriniferi with the products of decomposition and later on through secondary nephritis (*Hofmeyer*, *v. Mering*). The most important toxic change in the composition of the blood is, according to *Marchand's* opinion, to be sought for in the metamorphosis of hæmoglobin into methæmoglobin, a product totally useless for interchange of gas. The dispute which has arisen latterly between *Stokvis* and *Bokai* on the one side and *Marchand* on the other side, whether blood poisoning really occurs or whether the cardiac paralytic effect of chlorate of potash does not play the chief part, may be considered as decided in favour of the last-named author. He has proved that the negative results in rabbits are caused by the animals being too quickly killed through gastroenteritis toxica. He has also shown (a fact already proved by *Lenhartz*) that one finds the characteristic methæmoglobin in dogs' blood (according to *Cahn*, accompanied with sugar). We have also been able to prove the same in human beings, and as rapid as the effect of the poison might be, never have we missed the characteristic alterations in blood and kidneys, in opposition to *Leichtenstern* who only speaks of cardiac paralysis. Chlorate of sodium, as well as other chlorates, produces equally serious symptoms as chlorate of potassium.

2. *The toxic effect of specific disease.*—To this class belongs hæmoglobinuria in the course of serious general illnesses, particularly acute infectious diseases, as typhus (*Immermann*), scarlet fever (*Heubner*), diphtheria (*Küster*, *Salkowski*), small-pox, septic fevers, scorbut, intermittent fevers (*Stolnikow*, *Sorel*), also the secretion of hæmoglobin as a symptom of *Winckel's* disease (cyanosis neonatorum icterica perniciosa).

3. Injection of *water* and of salt solutions, the concentration of which varies according to the different animal-species, has a

dissolving effect on the blood corpuscles (cythæmolytic), also glycerine (*Jitta*), and above all transfusion of heterogeneous blood; the serum of one species has a toxic effect on the other species (*Panum*, *Ponfick*, *Prévost-Dumas*, *Landois*, etc.).

4. Hæmoglobinuria can result through *thermal* irritation, such as lesion by lightning (*Obernier*), particularly as a sequel to extensive burns. The red blood corpuscles are dissolved by the excessive heat from which the blood suffers in circulating through the burnt parts (*v. Lesser*). Sometimes a genuine nephritis ensues (*Falk*, *Wertheim*).

5. Hæmoglobinuria has been described as a *fat embolism* (*Scriba*, *Riedel*).

6. Finally a spontaneous hæmoglobinuria of an undecided origin sometimes occurs, which is indisputably connected with *syphilis* (*Schumacher*) and *malaria.* The most interesting clinical form is the periodical (*Lichtheim*), paroxysmal (*Küssner*), intermittent hæmoglobinuria. H. a frigore (*Mesnet*), da freddo (*Murri*) Winterhæmoglobinuria (*Hassal*), psychrogenic (*Boas*) rheumatic hæmoglobinuria, hæmoglobinuria produced by over-walking (*Fleischer*, *Strübing*, *Kast*, *Bollinger*, *Robin*).

Fröhner has established a parallel form in horses. The same is, however, not always produced by the effects of cold and muscular exertion, but rather caused by degenerative processes in the muscles. During the attacks urea and muscular pigment are found in the blood. Decreased resistive power of the red blood corpuscles and abnormal small affinity of the hæmoglobin with the stroma (*Leube*), from which it recedes, are said to be the primary origin. It is most probable that in cases, where the paroxysms ensue after a long walk, the products of fatigue after muscular exertion are of great influence (*Kast*). The corresponding theory of *Ralfe* and *Grainger Stewart* only agrees to a small extent with the real facts. Formerly it was an accepted fact that this illness left the kidneys as such intact, now several authorities, chiefly French ones, declare an anatomical lesion as possible; in fact, a renal origin of the illness (*Robin*, *Desnos*, *Millard*, *Bucquoy*, *Hagen*, *Prior*). As a matter of fact we can also corroborate that an intermediate stage exists between hæmoglobinuria and renal paroxysmal hæmaturia (*Dickinson*, *Saundby*, *Herringham*). This rare occurrence, however, in no way justifies us to adopt the theory of a primary renal lesion in paroxysmal hæmoglobinuria.

7. Hæmoglobinuria is in a number of cases caused by *parasites*, such as filaria sanguinis (*Mackenzie*), rhabditis (*A. Baginsky*), in the blood and in the genito-urinary passages. The attacks are generally confined to the night (compare "Parasites of the Kidneys"). The dangerous hæmoglobin in oxen in Roumania is of bacillary origin (*Babes*).

Pathological anatomy.—The kidneys are generally more or less enlarged and hyperæmic, of a brownish-red colour, are streaked in correspondence with the tubules, in consequence of brown granulated masses of hæmoglobin. These masses have reached the cavity of the tubules partly through the tubular epithelium (*Lebedeff*), and form, together with exudata, genuine renal casts. Ensuing the excretion of hæmoglobin and poisons, parenchymatous alterations (tumefaction, fatty degeneration, formation of vacuoles, necrosis of coagulated epithelium), and according to *Afanassiew* genuine glomerulo-nephritis with interstitial cellular infiltration, manifest themselves in serious cases (see part on "Nephritis"). The last-mentioned authority has most minutely studied and proved the changes which result from infusions of blood, from intoxication with toluylendiamin, with glycerine and pyrogallic acid, particularly with regard to the form of corpuscular destruction (coloured granules, granular segmentation) and the relation of hæmoglobinuria with icterus. He further proves that the dissolved blood and bile pigment is excreted by the glomeruli and the formed elements by the tubuli uriniferi. In cases where icterus is present, casts of hæmoglobin as well as such of yellowish-green bile pigment are to be found. In acute and specially per-acute cases of intoxication with chlorate of potash, bluish and grey colour of the skin and chocolate-brown colour of the blood (methæmoglobin) is remarked; in such cases the kidneys do not show any striking alterations. Nevertheless we have seen these organs dark violet, brownish, discoloured and completely obstructed with brownish-red coagulated masses of hæmoglobin, and the skin intensely cyanotic. All these symptoms can even appear when the toxic effect has scarcely lasted more than twenty-four hours. Serious diseases of the kidney must generally be excluded as primary cause of paroxysmal hæmoglobinuria; post-mortems have furnished but little information or such of minor importance.

Symptomatology.—The toxic and infectious forms of hæmoglobinuria often coincide with the general phenomena of intoxication. A description of these symptoms, as well as those produced by serious combustions, belongs to a later part of this book. We must add that in cases of intoxication with chlorate of potash sudden dyspnœa, a filiform pulse, icterus and cyanosis and even serious cerebral derangements with coma often develop themselves. Death can take place in a few days from anuria. Examination of the blood sometimes shows peculiar changes of form of the red blood corpuscles (*Riess*) and acid reaction (*Friedländer*). In one case of permanent hæmoglobinuria, observed during a case of intermittent fever, *Heinemann* could not even discover one single normal blood corpuscle; although the corpuscles were all in a discoloured condition and floated together with masses of granules in the red plasma, life was not extinct. According to the severity of the cases, hæmoglobinous urine shows different coloration, varying from light red to dark brown or port-wine colour. It is difficult to discriminate whether the concomitant oliguria and anuria is to be considered as caused by obstruction of the tubuli uriniferi with masses of hæmoglobin (*Ponfick*), or whether it is the manifestation of intense adynamia (*Cohnheim*). In slight cases morphotic elements are seldom to be found; after standing, the urine deposits an abundant dirty-brown sediment, which, however, does not contain blood corpuscles, but detritus and yellowish-brown confluent masses of a peculiar shape, which *Cohnheim* most significantly compares with myeloid forms. These very same masses furnish the material for the above-named hæmoglobin casts, which are often to be met with in considerable number and size in the urine. Numerous brown particles, formed in the urine passed in the last stage of a case of fatal poisoning with chlorate of potash, attracted our attention; microscopical examination proved these to be a conglomeration of the products of disintegration of the multiform discoloured red corpuscles and pigments. One also occasionally finds hæmatoidin (*Strübing*). The excretion of solids, especially that of urea, being suppressed, specific gravity is usually abnormally low. The spectrum manifests bands of oxy-hæmoglobin, and methæmoglobin, but more seldom of hæmatin. In cases complicated with jaundice the urine is of an olive-green colour, urobilinuria (*Gerhardt*, *Gubler*, *Dreyfus-Brissac*).

Paroxysmal hæmoglobinuria.—We will now give a short clinical sketch of paroxysmal hæmoglobinuria, a disease much discussed latterly. Cases of this disease are scarce and rarely seen in hospitals, the general health being seldom permanently affected by it. Among the chief predisposing causes, cold stands foremost on the list (especially in winter), then severe muscular effort and strain, as after long marches, now and then psychical agitation (*Küssner*), abuse of alcohol, venereal excesses (*Lépine*) and menstruation (*Wolff*). Finally, every apparent cause can be wanting (*Ilgner*, *Lépine*). *Morris* noticed attacks which appeared regularly in weekly intervals. This disease has a preponderance on the side of young adult males (*Saundby*); the disease is probably hereditary; immediate connection with scrophulosis, rheumatism and gout is somewhat doubtful; on the other hand, connection with syphilis and intermittent fever is doubtless. The paroxysms manifest themselves as follows: the patients yawn, have drawing pains in the limbs, headache accompanied with nausea and become livid. These precursory symptoms are followed by a chill, or even intense rigor, then by fever and sweating. The patients are then in a state of nervous exhaustion and pass deep red hæmoglobinous urine, and usually have an icteric tint. The urine then following is of lighter colour, and in twenty-four hours the whole attack is over. Only now and then general debility, dyspepsia, pains in the liver and tumour of the spleen last for days. Rigor, fever and perspiration are symptoms seldom missing. Occasionally the attack is accompanied by urticaria and even by cutaneous œdema. The attacks generally commence and end with slight albuminuria (*Kast*). *Rosenbach* regards the precursory transudation of albumin as a symptom of the attack, an opinion shared by English physicians. The paroxysms have been artificially produced by simply putting the hands in cold water (*Weber*) or taking cold foot-bath (*Rosenbach*). During the paroxysms the serum of the blood is of a ruby red colour (*Küssner*, *Du Cazal*). *Ehrlich* constricted the fingers of a person suffering from hæmoglobinuria with an elastic ligature and then immersed the same in cold water; dissolution of red blood corpuscles and total discoloration of the stroma was the result. It is evident that the dissolution of the blood corpuscles is only produced in the parts exposed to cold; and from thence the products of disintegration pass into the residuary blood. A genuine renal disease must be excluded. Formed elements pointing to nephritis do not exist during the attacks, and in the intervals the renal excretion is generally normal. The sum total of the symptoms forms probably a state analogous to uræmia resulting from the secretory insufficiency of the kidneys, which is caused by the accumulation of the derivative products in the disintegration of blood in the tubuli uriniferi. The primary reason for the decrease of the resistive power of the blood corpuscles is uncertain. *Rodet's* theory of the increased excitability of the vaso-constrictors explains but little.

Treatment.—The treatment of hæmoglobinuria caused by intoxication and by combustion is naturally identical with that of the primary complaint. Transfusions are frequently recommended against intoxication produced by chlorate of potash. We are of the opinion that serious cases are incurable, and that slight ones get well of themselves. Dangerous weakness of the heart having once set in, all stimulative remedies are, according to our experience, not of much avail. It is decidedly dangerous to treat the "uræmic" symptoms with purging medicine, hot baths, pilocarpine, as is often recommended. Sometimes moderate bleeding followed by subcutaneous injections of chloride of sodium is of use. To render the blood more alkalescent by large doses of carbonate of soda is sometimes advantageous (*v. Mering*). Unfortunately the stomach of the patient usually revolts at this treatment. The treatment of the tenacious intermittent forms of hæmoglobinuria can be a prophylactic or a causal one. The prognosis cannot be considered favourable, as death often occurs from intercurrent illnesses (*Wolff*), specially phthisis; young children generally succumb to the first attack (*Demme*). *Saundby* was the first who advised a sojourn in a warm climate; we can only corroborate his opinion. If there is any history of malaria or syphilis we naturally administer quinine, iodine and mercury. Decided success is, however, rarely met with; but energetic anti-syphilitic treatment has sometimes excellent results. Three instances came under our observation which are entirely and permanently cured. *Murri*, *Schumacher II.*, *Götze*, *Kopp* and *Ingria* give like evidence. Hygienic measures are of the greatest importance, their principles are very simple.

IV. RENAL CASTS.

These morbid products were first clearly described about forty years ago by *Henle*. They are of most important clinical evidence; the proof of their presence can be even of greater moment than that of albuminuria. With regard to their presence in the urine our own wide experience enables us to make the following propositions:—

1. *Casts are connected with renal albuminuria in the words' widest meaning.* Although these elements are sometimes to be found in non-albuminous urine, this fact is in no way modified, inasmuch as casts can appear as precursory or consecutive manifestation (*Rosenstein*); or even it may be possible that our usual methods for testing are insufficient to prove the presence of the latter.
2. *The presence of renal casts does in no way of itself prove an anatomical lesion of the kidneys.*
3. *Renal casts are frequently absent in most serious cases of renal disease.*
4. *Their pathognomonic value depends on the variety of their formation.*

Distinct methods for diagnosing the presence of renal casts are to be found in manuals on the analysis of urine. The simplest and surest way is to allow the urine to settle for several hours in a conical-shaped vessel; the necessary quantity of deposit is then withdrawn by means of a pipette. If no sediment subsides, water should be added (as also when the urine has become hazy through urates of after ebullition), or the urine is decanted and the remaining small quantity placed in a filter of the finest smooth blotting-paper. The residue which clings to the paper is carefully put on the object glass of the microscope.* But only a few of the classifications of renal casts actually adopted have been sufficiently studied. The well-known and accepted one of *Bartels* tears affinity asunder and unites diversity. He separates "epithelial utriculi" and "blood casts" from genuine renal casts, and classifies hyaline and dark granular and waxy casts.

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* *Sehrwald* (*Deutsche Wochenschrift*, No. 24, 1890) seeks a reason for the so frequently observed fact, that casts disappear for a time out of the urine of nephritic patients. *Bizzozero's* explanation, that the reason is to be found in the fact that the casts adhered longer inside the kidney, can only be accepted at the very commencement of albuminuria. *Sehrwald* found by testing the urine every hour in a case of parenchymatous nephritis that in the course of the day the quantity of casts varied considerably. This difference cannot be imputed to the activity of different renal regions, as then the urine, containing but few casts, must also have less albumin and more water, which is not the case. In such cases where only few cylinders were to be found, they were strikingly pale and delicate, but had heaps of granules and loose nuclei, thus

As our knowledge stands at present, it is very difficult to establish a rational classification which includes all the known forms. *Leube's* classification, slightly modified, seems to us to respond the best to the clinical facts and to the wants of the practitioner.

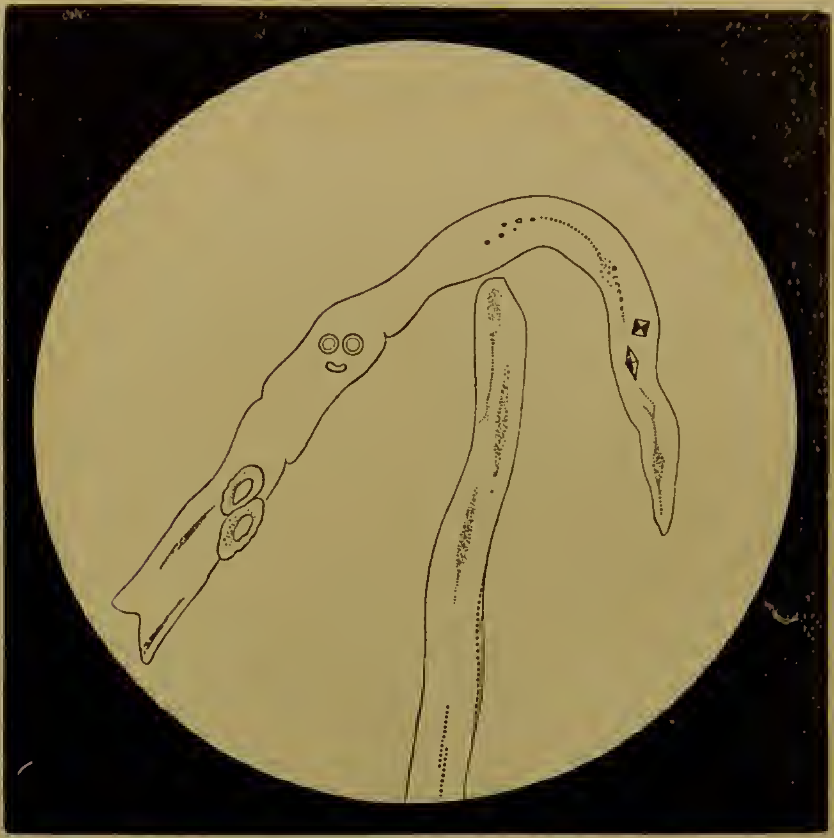

Fig. 1.

The term "fibrin casts" is best avoided, as many forms thus designated do not consist of fibrin, but of an albuminoid substance (*Rovida*) and an unexplained kind of protein (*Knoll*).

appearing as a fresh disintegration of the casts in the urine. As simple chemical solution is not obvious, and putrefaction to be excluded, the idea struck *Sehrwald* that the pepsin contained in the urine might produce this dissolution. After various experiments he found that in nephritic urine the decrease of pepsin was parallel to the decrease of casts. The practical deduction follows that in testing for casts one must neither leave the urine too long in the bladder, nor let it settle too long after being passed. *Sehrwald* believes that the peptic power of urine begins already in the kidney, and when the casts adhere in the tubuli uriniferi it loosens them and aids their excretion.—W. H. G.

Apart from *Vogel's* blood casts, the constituents of which are merely red blood corpuscles and fibrin (they being thus simple hæmorrhagic products of coagulation), we distinguish three principal forms :—

1. *Soft hyaline or homogeneous diaphanous casts.*
2. *Simple epithelial casts.*
3. *Metamorphosed casts,* which include :—
    - (*a*) *Granular casts ;*
    - (*b*) *Hard hyaline or waxy hyaline casts.*

One cannot insist too much on the fact that numerous mixed and transitional forms exist.

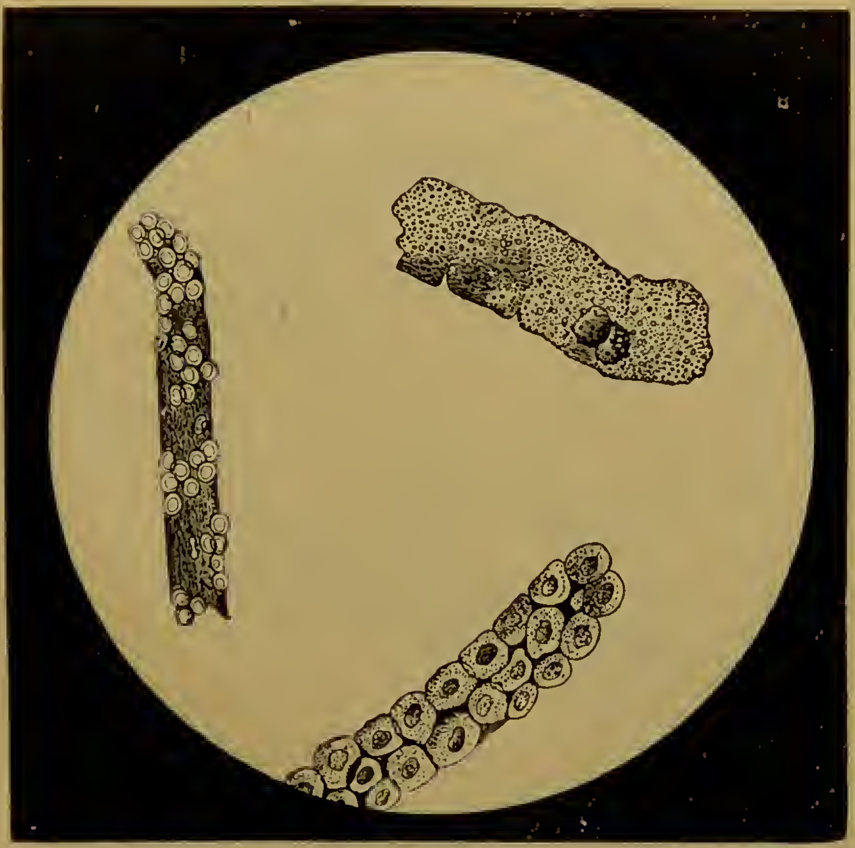

Fig. 2.

1. *Soft hyaline casts.*—These casts are colourless, diaphanous, homogeneous, sometimes slightly striated and dotted (detritus, fat), are soft, elastic, narrow (about 0·01 μ.) formations of various length (seldom longer than 1 μ.) (fig. 1). It is often exceedingly difficult and indeed sometimes impossible, if the diaphragma of the vizor of the microscope is large, to distinguish the outlines of these glassy renal casts. They are easily discovered if stained with carmine, chromic acid, with aniline colours, and particularly with iodine and picric acid (*Bizzozero*). The best tinctures are produced by bile in icteric organisms. The long forms are more frequently found than the spiral, shrivelled ones. Considerable reductions

and bifurcations are exceptional. If the urine be alkaline these casts disappear very rapidly.

2. *Simple epithelial casts* are disintegrated epithelium of the tubuli uriniferi (desquamative casts); they are probably not alone simple tubes (epithelial utriculi) but have as basis a hyaline substance (*Reinhardt*, *Wagner*) (fig. 2).

3. *Metamorphosed epithelial casts* are connected with the preceding kinds by numerous transitional forms. The epithelial constituent is effaced, the outlines of the cells and nuclei are indistinct or entirely obliterated, or replaced by granular products of disintegration. The metamorphosis thus far developed, we meet with granular, dark granulated, friable and cleft casts, which are often of a brown colour through the adherence of pigment. If further metamorphosis takes place the granular substance can become glossy, homogeneous and hyaline; these hyaline formations, however, distinguish themselves in a most significant manner from the already described soft colourless casts; they are compact, resistant, refractive and of a yellow colour, and are therefore rightly named glossy, waxy casts (*Bartels*). In two cases of serious chronic hæmorrhagic nephritis we have found numerous types of yellowish-brown casts, and occasionally quite

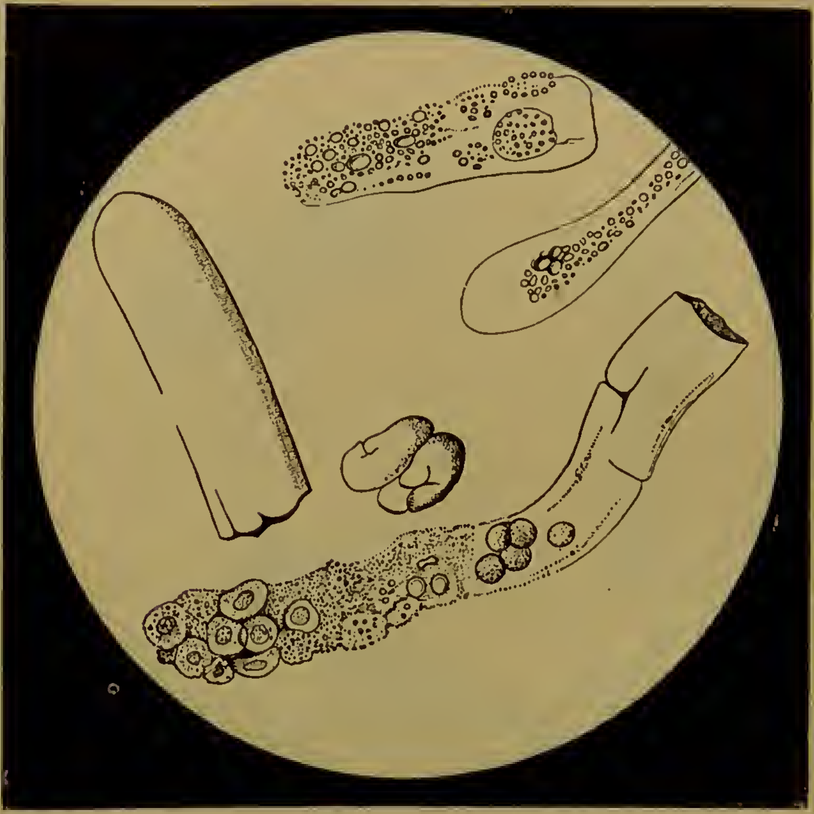

Fig. 3.

colourless ones (*Bizzozero*). Amyloid reaction seldom takes place. On minute examination we frequently find partly granular, partly waxy hyaline forms, and individual forms show us in a most argumentative manner the successive transitional stages from the apparently intact epithelium through the granular products of disintegration to hyaline. Waxy, glossy, cellular products and larger flakes often accompany the waxy casts, which are generally remarkable for their greater breadth. *Bizzozero* forms combinations of soft hyaline and waxy casts; we have repeatedly met with them of late (fig. 5). These casts, styled by *Löbisch* as finely granulated, and which often appear dotted with hyaline constitution, belong for the most part to the first category, rarely to the group of metamorphosed casts. Discrimination is sometimes very difficult. The same can be said of peculiar mixed forms of fatty granular casts with hyaline elements, forms with which we have frequently met several times. All casts can possess extraneous aggregation, such as blood corpuscles, epithelium, various cellular debris (inasmuch as they are not to be considered as integral components of metamorphosed casts), fat granules, fat globules in compact groups (fatty casts) (fig. 3), fat crystals.

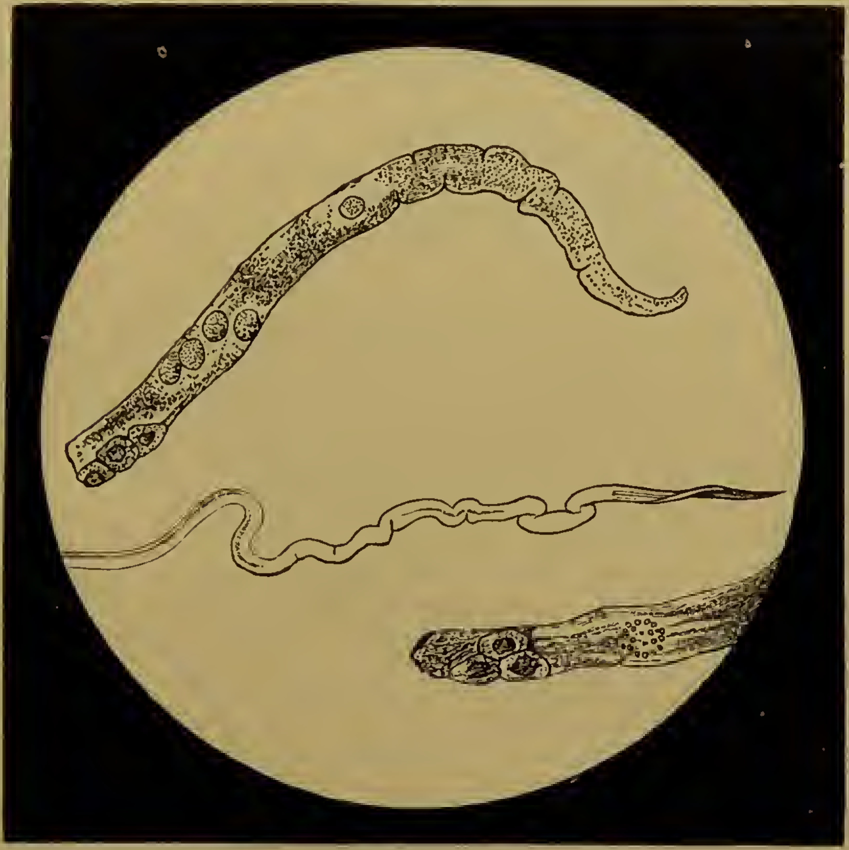

Fig. 4.

(*Knoll*), amorphous crystallised precipitates (urates, uric acid, oxalate of lime, etc.), finally micro-organisms.

Casts of abnormal shape are pretty often met with; infracted fibrillated, dichotomic (*Vierordt*), special curved (*v. Jaksch*), involuted, faceted and pronged. If of considerable length, filamentous and filiform, they constitute cylindroids, the thin varieties of which were first described by *Thomas* (fig. 4). They sometimes resemble mucus plugs, which leads one to doubt their renal origin. We have met the most decided transitional forms between ordinary hyaline and finely granulated casts, and agree with *Rosenstein* in not attributing a particular value to the discrimination or importance of these formations.

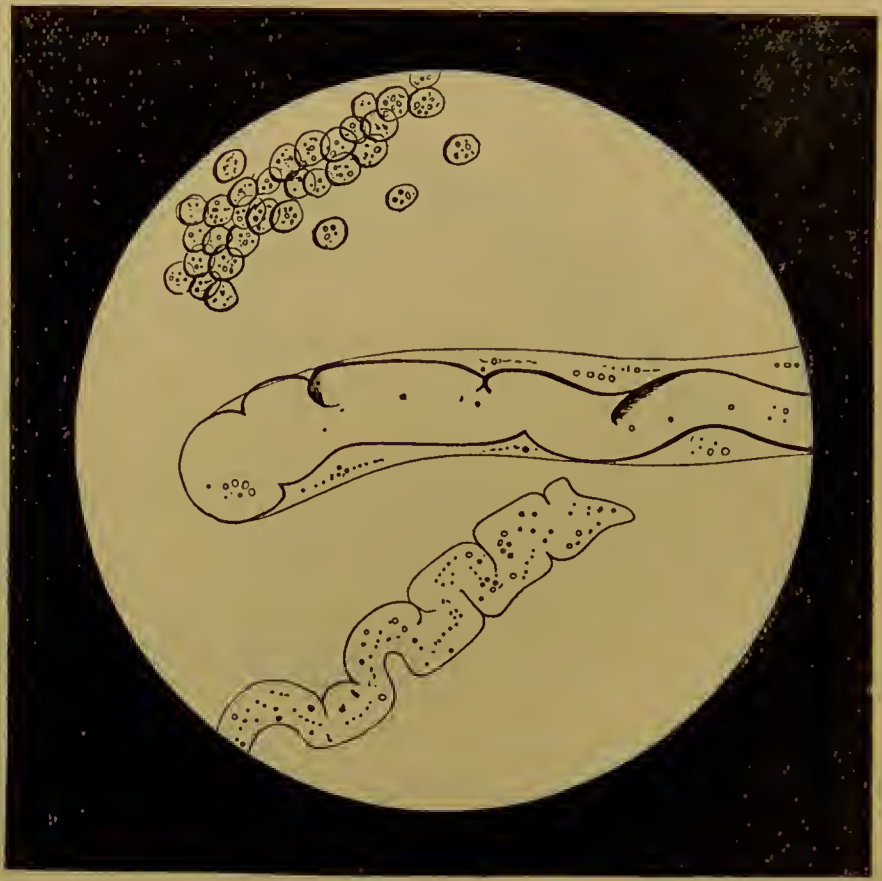

Fig. 5.

Pseudo-casts (non-organised casts, *v. Jaksch*) can easily be mistaken for genuine renal casts; the same mistake can also be caused by aggregation of urates, by plugs of bacteria (*cf.* septic nephritis and pyelonephritis), by conglomerations of hæmoglobin already mentioned,—it must, however, be remarked that mixed forms of this type with genuine casts also exist,—by mucus plugs (mucous casts), which are remarkable for their longitudinal plicæ, for non-parallel edges and great length, by secretion of the seminiferous canals and of the prostatic gland. The micrococcus casts (*Martini*) are delicately and symmetrically dotted, and can be instantaneously recognised by the resistance they offer to liquor potassæ.

Origin of renal casts.—How blood casts originate has been already fully discussed. In spite of numerous clinical and experimental researches consensus does not exist. We must consider the tubuli contorti as the main place of formation. The contorted forms, however, are not conclusive for this cast. Their frequent appearance in the loops generally proceeds from their difficult passage through the tubules. Experimental evidence proves that renal casts arise from compression of the renal vein (*Burkart*, *Weissgerber* and *Perls*, *J. Singer*, etc.), from the temporary ligature of the renal artery (*Litten*, *Posner*, *Ribbert*), from obstruction of the ureter (*Aufrecht*), by intoxication with chromic acid, combined with coagulative necrosis of the epithelium (*Gergens*, *Weigert*, *Kabierske*), from intoxication by cantharides (*Schachowa*), etc. Thus it is evident that renal casts do not always originate from the same cause; the epithelium in some cases plays a part, in other cases none.

It must be considered a fact that *hyaline casts* can be formed by coagulation of albumin, which transudes out of the glomeruli into the tubuli uriniferi. According to *Weigert* the coagulation results from the dissolution of cellular elements degenerated by a fibrogenous fluid, through the co-operation of which white blood corpuscles are likewise transuded. The epithelium remains intact, as *Singer* recently confirmed by means of experimental phlebostenosis. *Ribbert* cleverly proved the fact by intravenous injections of carmine; the colouring matter was excreted by the glomeruli and appeared not only in the albumin of the capsulæ, but also in the casts, but not in the epithelium. According to this exponent, transformation takes place under the influence of acid reaction of the renal parenchyma. *Posner* also recognises in the albuminous exudata the most probable source for formation of casts.

The *metamorphosed* casts are chiefly formed in the tubular epithelium. The degenerated glandular elements replace the white blood corpuscles with coagulable lymph, which proceeds from the glomeruli or from the tubuli bared of their epithelium (*Weigert*); this formation is analogous to the croupous processes. The hardened epithelium becomes disintegrated and forms a granular mass, which later on becomes hyaline and frequently undergoes waxy degeneration, or the epithelium forms homogeneous products of glossy transformation which become fused (*Burkart*, *Rindfleisch*, *Bartels*, *Aufrecht*, *Langhans*, etc.). *Singer* has seen casts form themselves in such a manner that the transudation is divided between the tunica propria and the epithelium; the latter becomes degenerated and forms the nucleus.

This observation explains the mixed forms observed by *Bizzozero*, *v. Jaksch* and ourselves. Co-operation of the red blood corpuscles produces the *pigmented* casts, which again change colour. *Bizzozero* suggests that the casts are also coloured by the urine. We consider it somewhat questionable that casts should be formed solely out of hyalinised colourless blood corpuscles, rather we believe they are often confounded with epithelial cells resembling leucocytes. On the other hand one frequently meets with cylindric conglomeration of intact leucocytes (fig. 5).

Thirdly, *casts of exudation* are described, *i.e.*, of exudative products from the tubular epithelium in the form of globules and plasma (*Key* and others). According to recent researches, however, these globular formations are also found under normal circumstances in the tubuli uriniferi. *Weigert* excludes active co-operation of the epithelium, but *Kelsch* and *Kiener*, *Lebedeff* and *Knoll* have to a certain extent taken up *Key's* theory.

With regard to the special *clinical importance*, it is not admissible to draw from the number of the casts decided conclusions as to the nature and kind of the anatomical lesions of the kidneys. Nevertheless their quantity is on the whole proportional to the albumin, and is at its height in chronic nephritis with heavy losses of epithelium. At the same time a diagnostical basis cannot be formed from the dimensions of the casts (*Johnson* considered their presence as particularly unfavourable for the case). Many broad casts were originally of cortical formation. Lessened diuresis and somewhat chronic morbid processes in the kidney are generally accompanied by the broad type of casts. *Cohnheim* denies the fact that the casts which one finds in the tubuli uriniferi are able to obstruct the passage of urine. According to the author's own histological observations it often happens that the lumina of the tubuli uriniferi are obstructed by the casts and the result is naturally an obstacle to the passage of urine. Small quantities of delicate hyaline casts are often found as a symptom of the so-called physiological albuminuria, which is a sequel to circulatory derangements. Their presence in these cases is of a harmless nature. Clinical experience accords here with the experimental (*cf.* the part on "Albuminuria"). At any rate as already remarked hyaline casts are not in themselves confirmatory of inflammation. *Nothnagel* noticed their presence with a case of intense icterus together with transudation of bile acids into the non-albuminous urine. But we must draw special attention to the fact that the contrary is not the case, and one must not believe that a serious disturbance of the kidneys is necessarily accompanied by intense formation of casts. Even in the worst cases of contracted kidney renal casts are often missing. On the other hand, the group of metamorphosed epithelial casts always indicates a decided disease of the kidneys, and especially inflammation. The more intact the epithelium is, the more likely is the existence of acute desquamative nephritis. It is not correct that the broad, waxy casts, as *Bartels* and various other exponents declare, are always confirmatory of a renal disease. Every year we remark these formations in one or another instance of acute scarlet fever nephritis, an illness in which *Tyson* and *Bizzozero* have also found them ("sometimes even a great quantity"). They are generally, however, indicative of a severe renal lesion and are always accompanied by other kinds of casts.

The aggregations are often of more importance than the casts themselves. They are the "forerunners of processes in the kidneys" (*Frerichs*). Thus numerous white blood corpuscles denote inflammation, moderate aggregation of fat globules as well as fatty casts manifest adipose degeneration of the parenchyma, red blood corpuscles announce the hæmorrhagic nature of the renal disease. According to *Johnson* small casts covered with leucocytes are a sign of glomerulo-nephritis. This assumption seems to us speculative (*cf.* part on "Nephritis").

V. DROPSY.

Symptomatology.—Dropsy is an important and characteristic symptom in many diseases of the kidneys and is often for the patient as well as for the physician the first indication of the seat of the disease. It especially appears as faithful companion of scarlet fever nephritis and the large white kidney. It generally follows in the course of albuminuria in a few days or weeks; nevertheless cases in which dropsy appears simultaneously or previously (precursory dropsy) are by no means scarce.

In the majority of cases dropsy sets in with *anasarca*, frequently in the face, especially in the eyelids, then also in the malleolar regions. In the latter case patients not confined to bed discover it in the evening, in the former when they awake in the morning. It is, however, characteristic of renal œdema that it changes without reference to its seat—in fact, springs from one place to another (*œdema fugax*). Dropsical accumulation in the genital regions, particularly in the scrotum, in the præputium and in the labia majora, is seldom remarked without concomitant swelling of the feet and legs. In cases of tumultuous progress the whole body can become unshapely swollen in a few days; the patient feels a sensation of tension and difficulty of movement in the extremities which becomes a great distress.

The manuals on physical diagnosis and general pathology must be referred to for description of the condition of the œdematous skin; sometimes in slight cases, specially in the face, pressure leaves no impress and the post-mortem does not reveal any other disturbance capable of explaining the anasarca. The careful physician will often notice (*Rosenstein* first drew our attention to this fact) dropsical swellings by exploring the crista tibiæ, signs which escape the notice of the patient and his surroundings; the sequel of serious anasarca (large vesiculæ, erysipelas, phlegmon, gangrene) can lead to fatal pyæmia and septicæmia. In acute nephritis it is not uncommon to find œdema without any infections of an inflammatory or even erysipelatous character.

Later on cutaneous dropsy is accompanied by such of the *serous* membranes, especially ascites (sometimes combined with stomachic and intestinal œdema), hydrothorax, and occasionally, but not always, hydropericardium alone. In chronic disease of the kidney, cutaneous dropsy of the upper part of the body diminishes when dropsy appears in the cavities. Transitional forms between simple transudation and inflamed exudata are particularly observed in cases of scarlet fever kidney (*Leichtenstern*).

The parenchyma of only few organs can be attacked by dropsy; the rapidly fatal œdema of the lungs is of the greatest clinical importance (regarding cerebral œdema *cf.* the part on "Uræmia"). To this type the interstitial œdema of the kidney itself belongs, the presence of which cannot usually be discovered even through the microscope.

Finally, it is not the mucus alone which is the seat of these symptoms; among others the œdema of the ary-epiglottic folds with its so much dreaded consequences. According to our experience these symptoms are not at all uncommon, as also the pharyngitis, to which one formerly attributed but little importance, and which frequently produces complications.

With regard to the chemical qualities of the transuded dropsical fluid, most excellent analyses exist, most of which agree with the theory of hydræmic serum sanguinis. The quantity of albumin found by *Runeberg* in the transudation ranges from 0·03-4 per cent. The quantity of urea varies according to the amount contained in the blood. It ranges from minute quantities to 1 per cent.; higher percentage is improbable.

The increase and decrease of dropsy is generally in reverse proportion to the quantity of urine passed, as is proved by *Bartels*' numerous and exact observations on diseases of the kidney. Exceptions to this rule are, however, very frequent. Dropsy can develop itself during plentiful diuresis; on the other hand it does not always appear simultaneously with anuria, even if the same be of several days' duration.

Pathogenesis.—The pathogenesis of renal dropsy is up to date not satisfactorily and fully explained.

The latest evidences speak against the earliest hypothesis, established by *Bright*. According to this observer, hydræmia (hypalbuminosis) in persons suffering from kidney disease renders as sequel to albuminuria the capillaries more permeable for the transudation of water. Latest experience, however, teaches us that diluted blood transudes by no means easier through the intact capillary walls than such of normal constitution (*Cohnheim*); furthermore that excessive loss of albumin, which the blood may suffer in healthy individuals through venesection and hæmorrhages in general, does not produce dropsy; on the other hand that in spite of inconsiderable loss of albumin serious œdema can quickly occur in acute nephritis.

According to the theory of retention of water (*Stewart*, *Bartels*) the want of albumin in the blood does not in itself cause dropsy; but the hydræmic plethora, *i.e.*, hypalbuminosis in connection with polyæmia. This hydræmic plethora is caused by the blood excreting decreased quantities of water and increased quantities of albumin. But there are many arguments against this theory, independent of the not yet decided proofs of an increase of the volume of blood of dropsical persons suffering from kidney disease. Firstly, one has noticed a peracute appearance of dropsy after scarlet fever, cold, etc., at a time in which hydræmic plethora cannot possibly have developed itself; further we have seen that the connection which *Bartels* established as constant between diuresis, albuminuria and dropsy is far from always being correct. One has seen patients attacked by obstruction of both ureters remain for a whole week without passing urine and without having shown the least œdema. It follows that, instead of admitting that dropsy increases and decreases according to the quantity of secreted urine, one could equally well maintain that it is the modifications of dropsical liquid which are primary, and that anuria and polyuria are only the sequels. Finally, *Cohnheim* and *Lichtheim* have shown that in experimental hydræmic plethora, produced by ample infusions of solution of chloride of sodium into the vascular system of animals, ascites, dropsy in the abdominal organs, in the salivary glands, with effusion of the fluid through the secretion of the organs, are produced, without œdema appearing, while, on the other hand, renal dropsy at first attacks the skin and spares the serous cavities. This points to abnormal permeability of the vascular tunica in consequence of local skin disturbances. This explanation of cutaneous dropsy by means of vascular changes is not new. *Frerichs* already mentions in his monography a paralysis and increased porosity of the capillary tunica as one of the causal circumstances of dropsical exudation. Furthermore, œdema was produced by experimental lesion of the spinal cord, in consequence of extinction of the vascular tonus (*Gergens*). *Cohnheim* and *Lichtheim* have made experiments which throw a decided light on the pathogenesis of anasarca. They have remarked anasarca in hydræmic animals in the cutaneous regions where inflammation has been artificially produced. *Cohnheim* concludes from this, that functional disturbances of cutaneous capillaries are the immediate cause of dropsy, while hydræmia and hydræmic plethora are only indirect causes. In fact, where functional cutaneous derangements are considerable, altered condition of the blood is not a necessary assumption, as in certain cases of nephritis produced by scarlet fever and cold. But when these primary cutaneous disturbances are not evident or are only slight, then a prolonged hydræmia can in its turn produce lesion of the vascular tunica, and it is thus that anasarca in certain forms of cachexia can be explained.

Certain improvements on *Cohnheim's* theory have been made latterly by experimental and clinical observations; thus *Jankowski* argues that when the blood is in a hydræmic condition the action of the vaso-motor nerves alone suffices to produce typical œdema. Finally *Strübing* and *Widowitz* describe peculiar cases of acute œdema to which they have given the name "angioneurotic"; these cases are caused by increased irritability of the dilatators, and, in course of some cases, one has sometimes noticed permanent cutaneous alterations (*Riehl*).

Even *Cohnheim's* theory is not perfect, serous dropsy is generally to be met with in most cases of acute nephritis (*Wagner*); we ourselves have very frequently noticed in cases of scarlet fever nephritis considerable ascites and hydrothorax duplex without such cutaneous œdema (which increased later), and *Gärtner* has succeeded in producing in dogs, by slow injections of water into the vascular system, in conformity with *Magendie's* former experiments, excessive œdema. This observer declares the negative success of *Cohnheim's* attempts to be produced by the fact that through forced injection intense hydræmia is produced, which leads to rapid excretion of the surplus and does not allow comparison with hydræmia of persons suffering from kidney disease. *Francotte* confirms *Gärtner's* assumption; he produced in dogs œdema through infusion of considerable quantities of fluid, and proved at the same time, that in nephritic diseases of this species of animals, ascites first appears and subsequently anasarca. *Francotte* likewise proves that experimental cutaneous inflammation considerably favours dropsy by means of plethora serosa. On the other hand the effect of cold and experimental anæmia have no effect. A primary change of the skin is, however, not absolutely necessary. *Rosenstein* draws attention to the impossibility of the presumptive cutaneous inflammation in malarial kidney disease. In this form of disease dropsy is intense and frequently precedes albuminuria.

According to *Leichtenstern* dropsy after scarlet fever is in so far analogous with scarlet fever nephritis, as it is also produced by the reception of the scarlet fever poison which causes lymphatic engorgement.

Although it is from the foregoing apparent that a uniform theory of dropsy does not exist, retention of water and hypalbuminosis undoubtedly belong to the principal causes, and it is a matter of further investigation to discover whether they act alone or in co-operation. But evidently the cutaneous œdema in constituting transitional forms of inflammatory exudations, as in the case of scarlet fever nephritis, must be strictly excluded from the œdema which appears in chronic nephritis, œdema which is more rightly called cardiac, and which spreads over the lower extremities in the form of anasarca (*cf.* scarlet fever nephritis and contracted kidney). By declaring the last-named forms of engorged œdema to be dependent on circulatory derangements and on insufficient cardiac compensation, *Cohnheim* verges towards the old classification of dropsy, *i.e.*, acute inflammatory (hydrops irritativus) and chronic passive form (hydrops mechanicus). The best safeguard against dropsy is doubtless a compensatory cardiac hypertrophy; but when this does not exist diaphoresis, catharsis and emesis must be considered as excellent compensators, which sometimes are the means of a kind of natural cure.

Diagnosis.—The diagnosis of renal dropsy is, apart from its typical localisation, formed by testing the urine. We must, however, bear in mind that scarlet fever dropsies unaccompanied by albuminuria or nephritis have been observed (*Philipp*, *Quincke*). The absence of decided cyanosis in pure nephritic forms of dropsy is not unimportant. In complications with valvular disease or engorged albuminuria, diagnostical errors are not to be avoided. In cases of dropsy combined with retention of urine, which results from paralysis or stricture of the ureter, and in cases of anæmia, nephritic casts are seldom found, and even the presence of a remarkable quantity of albumin in the urine is an exception. The same may be said of the important type of "essential" (primary, idiopathic) dropsy, which is strikingly analogous to the cachectic form. This primary kind, which constitutes the most prominent form in infants, often escapes clinical and pathological observation. As *Wagner* states in his valuable monography, various etiological moments must be taken into consideration, such as sanguinary, vascular and cutaneous alterations and debility of the heart. Finally, the lately so much discussed forms of myxœdema can be mistaken for dropsy. This illness consists of infiltration of the subcutaneous intercellular tissues with a serous gelatinous substance, which penetrates into the deep strata and is analogous to scleroderma. Myxœdema is generally to be found in the face. The observations of numerous authorities (*Gull*, *Gordon*, *Riess*, *Reverdin*, *Charcot*, *Virchow*, *Erb*, *Ord*, *Horsley*, *Hallibarton*, *Semon*, *Hadden*, *Mosler*, *Manasse* and others) prove that a link between nutritive derangement ("cachexie pachydermique") and mental debility, between morbid destructive processes and operative ablation of the thyroid gland (cachexia strumipriva), exists. Dependence on chronic nephritis can be absolutely denied (*Grocco*).

It is possible that less typical forms of myxœdema are often taken for dropsical swellings. The same may be said of cutaneous œdema which has become permanent in consequence of frequent recidivation of erysipelas and of affinite cutaneous affections. This œdema, which is generally localised in the face, *Lassar* and ourselves have often observed in cases where there was not the slightest trace of nephritis. *Hartmann's* statement that he had produced dropsy and albuminuria in his own "sound" body by nourishing himself exclusively with ham and bread needs as yet a plausible explanation; the fact, however, cannot play any prominent part in the differential diagnosis of renal dropsy. The treatment of dropsy is to be found in the part on the "Treatment of Diffuse Nephritis".

VI. URÆMIA.

Uræmia (from *οὖρον* and *αἷμα*), urinous infection, typhus urinosus, is a collective name used to describe various functional disorders of the central nervous system, of the respiratory and digestive organs. It is met with in numerous kidney diseases (especially scarlet fever, contracted, puerperal, and cholera nephritis), and in illnesses of the urinary organs with obstruction, and also in cases of liver complaint (*Debove*). The frequency of uræmia varies considerably under the influence of climate. Thus, according to the observations made by *Bright*, *Christison* and *Gregory*, about half of the patients suffering from Morbus Brightii in England become uræmic, while in Germany and Holland one can hardly reckon on an average 25 per cent. (*Frerichs*, *Rosenstein*, *Strümpell*). In the medical wards of the Berlin Public Hospital (Friedrichshain), about 100 nephritic patients are admitted per annum; of this number we declare that about half show uræmic symptoms.

From a practical standpoint it may be advisable to retain *Frerichs'* classification:—acute and chronic forms of uræmia,—although transitional forms are not uncommon.

Acute uræmia appears both in acute and chronic diseases of the urinary apparatus. A precursory stage seldom fails, the symptoms of which are headache, giddiness, vomiting and mental apathy. The attack itself, whether it appears as sequel to such symptoms, or suddenly and unexpectedly, in the midst of the habitual occupations or conversations, generally takes the form of eclampsia or epilepsy. The patients become unconscious and are seized with convulsions; sensibility and reflex actions are lessened, and frequently cease altogether. The spasms, which in slight cases usually confine themselves to passing contractions of the muscles of the face or of the extremities, become more violent and general in serious cases. In the very worst cases the whole body is shaken with violent convulsions, respiration falters and becomes stertorous, the pulse becomes small and very rapid, the face puffy and cyanotic; patients foam at the mouth and grind their teeth, the fæces and the urine, if the latter is excreted at all, are passed involuntarily, the temperature rises to a considerable degree. Previous to the attack considerable retardation and irregularity of the pulse often sets in (*Thomas*, *Wagner*). During the paroxysms themselves we have, however, but seldom observed this characteristic, and cannot share *Rosenstein's* opinion, who declares an abnormal hardness and tension of the pulse to be a constant forerunner of the attacks, although this latter quality may be considered as the most frequent one. The reason of the increased temperature is not quite apparent, but, according to our experience, such a decided parallelism exists with the intensity of muscular exertion (spasms, jactitations) that this fact must be brought into connection with the uræmic fever. An exceptionally low temperature is very seldom observed during the attacks (*Strümpell*).

The chronic spasms only last some minutes. When only one fit occurs the convulsions cease gradually, and comatose condition succeeds. It is probable that this state is due to the pernicious effect produced by the deprivation of oxygen on the cortex cerebri (*Traube*). After a period of time, varying sometimes from a quarter of an hour to several hours, the patient awakes as out of profound sleep. As a general rule, however, the attacks return after a few hours or days with renewed vigour (we noticed over 100 attacks in three days, when death took place in a patient suffering from renal sclerosis) together with progressive stupor, which at last increases to permanent coma, out of which it is impossible to rouse the patient. This condition may outlast the foregone attack if death does not set in at the height of the paroxysm.

The chronic convulsions can be of a hemi-lateral nature (*Rosenstein*, *Strümpell*), and can be introduced or replaced by tonic spasms, followed by contractions of long duration (*Jaccoud's* "tetanic uræmia"), and even by trismus and tetanus (*Leichtenstern*). We have seen types of all the above-mentioned forms, combined with genuine tremulous agitation. There are cases, however, without spasms, where the acute uræmia appears solely as coma, as delirium (the patient giving utterance to screams and trying to jump out of bed, etc.) (*Thomas*); a child who came under our observation screamed in the most heart-rending manner day and night. On the other hand, uræmia can be observed without the slightest mental impairment.

Furthermore, we must mention *motor paralysis*, especially hemiplegia, as concomitant symptoms and sequels of acute uræmia. Recently various observers (*Pätsch*, *Charpentier*, *Leichtenstern*, *Jaeckel*, *Chantemesse*, *Tenneson*, *Raymond*) have drawn attention to such cases. Even in those cases where post-mortems do not give evidence of the existence of coarse anatomical lesions, we must not form a conclusive opinion as to the non-existence of organic changes, which, although being invisible to macroscopical investigation, may not be so to microscopical research. For further details on these facts and other analogous phenomena, doubtless connected with co-operation of certain localised parts of the brain, such as epilepsy (*Chantemesse* and *Tenneson*), hemianæsthesia, hemianopsia (*Brieger*, *N. Weiss*), aphasia (*Lancereaux*), a later chapter must be referred to.

The occurrence of *bilateral amaurosis* is more frequent than the last-mentioned disturbance, the ophthalmoscopic indications are generally negative, and the pupillary reflex is intact; the retina and the whole optical tract is therefore not affected. This attack of uræmic amaurosis, which can occur as sole symptom (*Ebert*, *Henoch*, *Rosenstein*), usually comes so suddenly, sometimes even in the course of a few hours, that a patient, whose vision before the uræmic attack was unimpaired, can awake out of the coma with total blindness. The derangement, however, generally takes a favourable course and prognosis. Uræmia attacks the *ear* much seldomer; these derangements, such as difficulty in hearing, deafness, singing in the ears, etc., generally come and go quickly.

Finally, acute uræmia can appear unaccompanied by any cerebral symptom, and be only characterised by serious derangement of the respiratory system and of digestion.

A retrospective glance at the various symptoms shows that the most frequent cases (as *Rosenstein* so decidedly argues) consist in *paralysis*, which generally attacks the sensorium and the sensory organs, and *irritation*, which mostly affects the motor tract.

Chronic uræmia, with its whole complex of symptoms, has found special and permanent description at the hands of *Frerichs*. It is often not recognised in the slighter cases, it steals upon the patients generally in a most insidious manner, and usually does not leave them; dull headache, violent migraine (*Bartels*), bodily and mental languor and inertia predominate. The patients become entirely changed; their relatives and physician hardly recognise in the expressionless and indifferent physiognomy the features of healthy days. While the lethargy increases the patients gradually fall into deep coma, sometimes interrupted with delirium; the co-operation of the motor tracts, however, is by no means seldom.

We must not close the description of uræmic cerebral symptoms without referring to an important link in their chain, to the *psychical* derangements. We have to thank the director of the asylum in Jena, Professor *Binswanger*, for the following communication:—

"The occurrence of *mental* derangements in the course of acute and chronic renal disease has been observed by various authors (*Hagen*, *Scholz*, *Jolly*, *Bartels*, *Wagner*, *Brieger*, *Raymond*, *Petrone*, *Kleudgen*, *Obersteiner* and others), and a causal union between both must be considered as coincidentally based on the already mentioned reasons. As yet valid, clear and convincing views on the physio-pathological origin of these psychical derangements are not to be found for all cases. The greater portion of the same is nearly related to acute uræmic conditions, and is best reckoned as belonging to mental derangements produced by toxic effects. In fact, the forms of mental derangement found in connection with the nervous symptoms of uræmia resemble to a great extent those which appear in the course of chronic alcohol, morphia and saturnine intoxication, in the course of intoxication with various gaseous, vegetable and metallic poisons. This resemblance of classical symptoms is particularly remarkable in those acute mental derangements which only last a few days, and which appear with eclampsia parturientium, and in the course of genuine nephritis, with or without epileptiform spasms. Apart from the loss of consciousness, the prominent symptoms consist in serious *hallucinatory excitations*, *terror*, excessive *motor irritability*, which attains in some pernicious cases the character of *delirium acutum*. These attacks of mental disturbance, which only last a short time, are less known, as they, in consequence of their acute character, do not come so frequently under the notice of the physician. In the lunacy wards of the Charité Hospital I saw six such cases, two of which ended fatally within a few days, and I then (1881) made a report relating to them in the Medical Association of the Charité. The observations found in medical literature have entirely to do with sub-acute and chronic cases, in which *stuporous* and *melancholy* conditions, also *hallucinations* with maniacal excitement, were described. These cases belong particularly to the type of *chronic uræmia* described by *Frerichs*, in which the symptoms resemble general psychical conditions of exhaustion. Also in these cases one must allow that renal diseases exercise a decided influence on the formation of psychical symptoms. This fact is proved by cases in which the mental disturbances began at the same time as the development of renal disease, and disappeared with the decrease of the same, as also those cases where, in the course of protracted renal disease, all exacerbations and remissions of the original disease kept pace with an increase or entire cessation of the symptoms of mental derangement. I had lately the opportunity of observing a case of the first-mentioned kind during a period of several months. About a month after kidney disease manifested itself, the patient, fifty years of age, was seized with attacks of terror, headache, 'buzzing and vacuity in the head'. Then insomnia, confusion, impossibility to concentrate thoughts, self-accusations, fictitious ideas of crime set in. The albumin decreases when the patient is treated with opium and calomel. Patient becomes quiet and mentally bright. A quarter of a year later violent attacks of terror with stupor reappear. Later on, the old complaint that the head is 'splitting' reoccurs, etc. The same treatment is used. In November no albumin in the urine, patient brighter and quieter. In the following months variable condition, but the appearance and disappearance of albumin always coincided with the psychical symptoms. When patient was dismissed all the symptoms had partially subsided, but were not quite extinct. From information received later on, I heard that the patient had recovered entirely.*

"From all the observations to be found in literature, and also from my own experience, I must say that the occurrence of decided mental disturbance in the course of renal disease is nearly absolutely connected with the existence of an *individual neuropathic predisposition*. The cases where mental disturbance lasts months after the acute kidney disease are difficult to explain (*Jolly*)."

Although uræmia most frequently attacks the nervous system, yet its effect on the *circulatory* and *respiratory* organs is very considerable. The number of respiratory derangements in renal disease is very great indeed. It is difficult, even almost impossible, to establish a decided differential diagnosis between the uræmic type latterly discovered, and described as *Lancereaux's* "cardio-pulmonary" uræmia, and that category produced by anatomical diseases of the respiratory and circulatory organs, as bronchitis, œdema of the lungs, and weakness of the heart of an organic and functional character (*Cohnheim*, *A. Fränkel*, *Leyden*). Only when such causal indications are missing, one may admit primary uræmic dyspnœa. Such is especially the case when asthma with free intervals occurs, manifesting itself in painful nocturnal paroxysms. This asthma resembles in clinical respect nervous bronchial asthma, which can appear suddenly, even as an initial symptom of renal disease, and *Cheyne-Stokes'* type (*Lancereaux*). In other less perspicuous cases permanent dyspnœa often exists with or without marked attacks, either with palpitation of the heart or without. The deep or superficial respiration is accelerated. *Leichtenstern* observed, and we must certainly agree with him, that dyspnœa is the most frequent symptom of scarlet fever uræmia. It is generally accompanied with great acceleration of pulse, pains in the cardiac and abdominal regions, and with violent jactitations. Post-mortems have frequently shown combinations of uræmia and dyspnœa in such of the cardiac, and especially myocarditic type; the patients had been mostly alcoholists or had suffered from arterio-sclerosis. Special types cannot be distinguished. When debility of the heart is caused by insufficiency of the heart muscle in consequence of kidney disease itself (*cf.* the part on "Contracted Kidney"), the distinction between cardiac and uræmic asthma is generally quite impossible.

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* I had occasion to observe a similar case. A patient, fifty-four years of age, had not been suffering from renal disease more than two months when, together with feeling of pressure in the head, mental derangement set in, such as melancholia, insomnia, loss of memory, excitement alternating with mental depression; conversation became disconnected and rambling. In the same measure as the quantity of albumin increased or decreased the patient's mental condition became worse or improved, till at last, when one succeeded to obtain relative freeness of albumin, all mental affections disappeared.—W. H. G.

An equally painful torture for persons suffering from renal disease consists in uræmic *vomiting*, which has been already mentioned as precursory symptom of acute uræmia. It continues for months, in spite of every dietetic precaution, and in spite of every kind of medical treatment. It must be considered as characteristic that in the beginning vomiting occurs in the morning, when the stomach is empty, and is accompanied with or without dyspeptic symptoms; the vomit consists then of mucous, serous masses. Sometimes, and especially in the later stages, the vomit has alkaline reaction, and betrays by its odour the presence of a large quantity of ammonia. *Lancereaux* has latterly described a kind of uræmia "pharyngo-buccale," characterised by the presence of clammy mucous masses, which accumulate in the mouth and pharynx. This classification seems rather far-fetched, if we remember the frequent combination of hypersecretory pharyngitis in serious renal disease. It must also be remembered that genuine gastritis, which often accompanies Morbus Brightii, can produce symptoms of deceptive resemblance to uræmic vomitings or other stomachic affections.

Uræmic diarrhœa, which is sometimes as tenacious as vomiting, has no specific character. An anatomical origin is nearly always to be found. Sometimes the gastro-intestinal uræmia, as *Déligny* specially emphasises, gives the patient the appearance of suffering from tuberculosis. If the patient be only under short observation false diagnosis is sometimes unavoidable.

Attacks of *singultus* must be reckoned to the symptoms of chronic uræmia; they generally pass quickly, but sometimes, however, they last for weeks, torturing the patient day and night (*Wagner*). Another symptom is the terrible scourge *pruritus*, which causes the poor victim, even in a comatose condition, to scratch himself in a frightful manner.*

Nature, pathogenesis, theories.—A number of experimental researches have been made on the question of the *nature of uræmia*, without a definite explanation having been arrived at. In spite of diversity of opinions a certain conclusion, however, cannot be denied.

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* To somewhat relieve the intense suffering of the patient I apply, with success, glycerine and lemon juice in equal parts.—W. H. G.

In order to characterise the relation of uræmia to that of urinary water, one must consider, as a rule, that the excretion of urinary water and solids is lessened so much that *anuria* sometimes arises. It is therefore natural that the cause of uræmia was sought for in the *retention* of uric constituents and derivatives in the system (chemical theory), in which, during the whole duration of uræmic symptoms, normal and even increased quantities of urine were excreted; this did not escape the notice of *Christison;* later on, these theories were undeniably proved by *Liebermeister*, *Wagner* and *Jakubowitsch;* on the other hand, anuria, without any kind of excretion, be it vomiting, be it diarrhœa, has been observed without the slightest sign of uræmia (*Payet*, *Biermer*, *Henoch* and others). Cases of the first category, which we occasionally notice, are apparently exceptions to the rule, inasmuch as great resorption can have taken place of a non-recognised œdema, or if a latent retention of solids existed, which can only be discovered by methodical and minute analysis (*Fleischer*), or, finally, the symptoms observed have no relation whatever to uræmia (*Thomayer* most precisely warns against confusion with alcoholism, cerebral tumours, etc.). Everything considered, one must not (especially with regard to cases of the second category) consider the relation of uræmia to the excretion of urine as always constant.

This last consideration, together with contradictory arguments which proceed from the chemical theory, have proved the necessity of a second hypothesis, by which the symptoms are explained from a different standpoint, by mechanical processes. *Traube* is the chief apostle of the "*mechanical theory*". According to him the basis of uræmic symptoms consists of *œdema, and as a sequel to it anæmia of the brain.* The œdema is produced through combination with hydræmia and high arterial pressure; some cause or other, especially where hypertrophy of the heart exists, is able to increase the latter and thus brings on serous transudation. The type of the uræmic attack depends on the localisation of the œdema in the brain; if the cerebrum alone is affected, only coma ensues, combined with convulsions as soon as the middle lobes are affected. *Munk* has attempted to experimentally support this theory, which belies the name uræmia; he actually succeeded in producing in dogs cerebral œdema with coma and spasms by means of injection of water in the carotis after ligation of the ureter and one of the jugular veins. *Rommeläre*, on the contrary, could not say anything in favour of the theory after a renewal of the experiments. Further, *Bidder* found that convulsions and coma could only be produced by injections when these increased notably the normal pressure in the brain. The *Cohnheim-Lichtheim* experiments show most decidedly that the brain of the animals experimented upon can remain dry, in spite of the incorporation of colossal quantities of liquid into the vascular system, and of the greatest possible increase of the blood-pressure. If, on the other hand, even clinical evidence proves that hypertrophy of the heart and hydræmia do not always exist with uræmia, then above all does the fact that cerebral œdema is an *inconstant* state in uræmia (*Bartels*, *Cohnheim*) prevent the universal acceptation of *Traube's* theory. (The argument that a negative post-mortem is a proof of the non-existence of œdema intra vitam is untenable.) In cases where cerebral œdema is found its presence may also be considered as a sequel to the spasms (*Bartels*, *Wagner*).

At the same time *Traube's* theory has still followers, and appears to be tenable in a limited number of cases. Apart from the undeniable advantages which the acceptation of a cerebral anæmia offers for the explanation of those cases of uræmia without oliguria, and of anuria without uræmia, *Rosenstein* draws attention to the fact that cerebral anæmia can occur in the most varied ways without œdema (for example, through saturnine intoxication). He inclines of late towards a kind of hypothetical combination of the chemical and mechanical theory, *i.e.*, that one or other of the retained toxic uric constituents principally affects the vascular nerves of the brain. *N. Weiss* accepts *Traube's* theory in a different form, inasmuch as according to his opinion a chemical agent cannot be causal in those cases where local cerebral derangements, which are indicative of a lesion, are inconstant (irritability, depression). *Leichtenstern*, however, decidedly declares scarlet fever uræmia to ensue from the action of infection, and in consequence of the sudden change of symptoms of anæmia and inflammatory œdematous conditions of the brain and its meninges. In a similar manner *Rühle* explains the uræmic attack to be produced by admixture of a toxic substance, formed in the blood itself, to the dropsical fluid ; according to him, cerebral œdema is a constant condition in persons who have died in uræmic attack.

To the *chemical theories* we must also reckon the hypothesis of an intoxication of the blood through uric constituents and their derivates, and their pernicious effect on the nervous system. Experiments demonstrate that animals die under sopor, spasms, vomiting and diarrhœa, when the secretion of urine is artificially restricted by ligature of the renal arteries, by nephrectomy, and by ligature of the ureters. Nothing was more natural than that one should make the chief constituent of urine, *urea*, responsible for this ; the quantity found in the different organs and fluids of nephrotomised animals was about in the same proportion as would have been excreted by the urine during an equal period of time (*Voit*). The presence of urea in the blood of persons suffering from kidney disease seems to harmonise herewith. But the attempt to produce uræmia by injecting solutions of urea into the circulation of animals was unsuccessful ; equally so were trials with other constituents, uric acid, creatin, etc. Such results caused *Frerichs* to make exact and profound experimental researches which resulted in his well-known theory that it is not urea, but *carbonate of ammonia,* which produces uræmic intoxication. The latter appears as product of decomposition of the urea accumulated in the blood under the influence of ferment which forms itself through varied derangements. But this theory only held ground for a few years, although the numerous important experiments and clinical observations of *Frerichs* seemed to support it and to divest the deficient parallelism between uræmia and urinary secretion of its wonders. A number of careful experimentalists (*Schottin*, *Hoppe-Seyler*, *Kühne*, *Strauch*, *Rosenstein*) found at the hand of more accomplished and precise methods, that a transformation of urea into carbonate of ammonia in the blood of uræmic persons does not take place, that the presence of ammonia in the breath is not dependent on uræmia, but on the presence of products of decomposition in the oral cavity of healthy and sick persons, and, furthermore, that intoxication with carbonate of ammonia produces symptoms resembling those of uræmia (*Petroff*). The minute quantity of carbonate of ammonia which *Spiegelberg* and *Gscheidlen* found in the blood in cases of puerperal eclampsia cannot be taken into consideration, and *Demjakow's* latest positive experiments are made on animals "uræmised with urine ferment". Even *Falk* only caused uræmic symptoms by injection of urine made septic by infection, not by spontaneous decomposition. Nevertheless, more recent observations have shown that in cases, where one has to deal with ammoniæmia, it is possible that the blood resorbs carbonate of ammonia from urea found in the intestines and urinary passages (*Treitz*, *Jaksch*).

A different arrangement of experiments seems to prove that it was by no means necessary to believe that a product alone should be able to produce uræmia, while we at the same time consider urea and other normal urinary constituents to be harmless. Thus in animals if one prevents the rapid elimination of urea by ligature of the ureters and extirpation of the kidneys (*Hammond*, *Gallois*), one will observe that in consequence of "overloading of the blood" with urea death will take place much quicker under uræmic symptoms than through nephrotomy alone. Other authors have not been able to prove this indisputably. *Fleischer* has latterly not been able to corroborate *Voit* and *Oertel's* view that under nitrogenous nourishment the symptoms of intoxication are steadily increased when the quantity of water introduced into the system is lessened, and decreased when it is heightened. Even if the blood is suddenly flooded with great quantities of urea, uræmic symptoms in dogs are often missing. The quantity of urea introduced by *Fleischer* direct into the blood was much larger than that which nephrotomised animals retained in the organism from the moment of operation till uræmic death occurred.

The fact that enormous quantities of urea are necessary to cause uræmic death has brought us quite recently to the idea of searching for other urinary constituents capable of producing intoxication. *Fleischer* infused 100 grammes of urea into the peritoneum of a dog, without rendering it uræmic, a fact which agrees but little with the calculations of *Gréhant* and *Quinquaud*, who assumed that subcutaneous injection of urea equal in quantity to 100th part of their body weight will suffice to kill dogs and rabbits. It must be considered as most important and interesting that *Feltz* and *Ritter* arrived after numerous and exact experiments at a result which entirely agreed with the experiments made by *Voit* and another experimentalist, *Astaschewsky*, who at the same time came independently to the same conclusion. The latter ligated the ureters of dogs and injected urea and mineral urinary constituents. A violent uræmic result ensued, more so from injections with salts of potash than from such of urea; thus he believed uræmia to be an auto-intoxication of the organism with an organic substance, particularly with salts of potash. The experiments of *Feltz* and *Ritter* on dogs with ligated renal vessels annulled the effect of ammonia, urea and a number of other organic urinary constituents, as also of extractive principles, which *Schottin* considered to produce uræmia, salts of sodium and potash, or only proved the toxic effect of a dose, which in reality is impracticable. Injections of water only produced simple increase of pressure (contrary to *Traube's* opinion), while an injection of inspissated urine deprived of its organic substances and of calcium salts had a fatal effect, an effect which proved all the more rapid the greater the quantity of salts of potash. In the blood of uræmic animals an increase of salts of potash was discovered. *D'Espine* was also able to prove an increase of salts of potash in the blood of persons suffering from scarlet fever uræmia. *Snyers de Liège* only found small quantities of the same in the blood of persons suffering from eclampsia. *E. Rosenthal* believes kalium chloratum to be the principal cause of vomiting, while *Rovighi* considers that salts of potash are one of the chief causes of respiratory derangements. Finally, *Landois*, *Leubuscher* and *Ziehen* have recently called attention to the extraordinary similarity of uræmia to the various chemical irritations which are produced on the cortex cerebri by creatin, phosphate of potassium, urates, etc.

Encouraged by *Bocci's* discovery of a special toxic constituent in the normal urine, a number of well-known French observers have made studies of the part played by this "urinary poison" as a uræmic principle. *Schiffer* had mentioned *Bocci's* substance as producing paralysis of nervous centres. This substance is doubtless analogous to the putrid alkaloids found by *Brieger* in artificial digestion of albumin. The symptoms of *Bocci's* substance exactly coincide with uræmia (depressed and irritative state). *Pouchet* and *Bouchard*, *Villiers*, *Feltz*, *Aducco* discovered various alkaloids in normal human urine, the quantity of which showed a certain dependence on the degree of organic oxidation (increased by hunger and fever, decreased by bodily exertion), and to which could be ascribed a prominent part of the toxic effect of normal urine (clonic and tonic spasms, coma). *Bouchard* has started a most ingenious but somewhat bold theory, which, however, is not yet sufficiently proved. According to it, a healthy person is a receptacle of poisons, which he receives and forms, the deleterious effect of which is usually prevented by the normal processes of excretion. The quantity of poison excreted in the urine in the course of two days would be sufficient to kill a person; it contains various toxic substances such as urea, potassium carbonate, with narcotic, spasmodic, mydriatic and other effects; the other chief excreting organ, the intestines, particularly carries away potash and ammonia. The retention of all these poisons matures the symptoms of uræmia as soon as the "urotoxic co-efficient" is attained. The urine of uræmic patients is *innocuous*, because its uræmic constituents are retained in the blood. Finally, *Doléris* and *Butte* believe to have found in the etheral extracts of the blood in eclampsia a crystalline substance, which has the power of uræmising dogs (*cf.* puerperal nephritis). Based on his experiments with animals, *R. Müller* has previously ascertained the vomitive and convulsive effect of the etheral and alcoholic extraction of urine.

An unprejudiced retrospective glance at the above-mentioned theories leaves no doubt that what we now-a-days term uræmia is a clinical collective name, and that an attempt to establish *one* sole cause as an explanation for all forms is, according to the extent of our present science, futile. A theory, explanatory of all the facts, can only be expected in the course of future attempts. The deficient accordance of the experimental results with *clinical* facts prevents a general theory. Uræmia is an "overloading" of the blood with urinary substances ensuing from a pathological disproportion between formation and excretion, therefore a form of auto-intoxication. Whether urea, potash salts or alkaloids play the chief part cannot yet be decided, and is also of secondary importance. Those conditions, which in animals are produced by nephrotomy or ligature or deprivement of water, occur in human beings through nephritis, inspissation of the blood in cholera and through various obstructions in the urinary passages. The more acute the causal moment, the more violent the uræmia; the slower the "overloading" of the blood, the more lingering the nervous symptoms. The prominent frequency of uræmic attacks in cases of chronic contracted kidney is certainly connected with hypertrophy and consecutive weakness of the heart through compensatory derangements (*Cohnheim*, *Leyden*). It is the cardiac insufficiency which leads to the decrease of the excretion of urine. The scarcity of acute uræmia in cases of genuine fatty kidney is explained by the fact that important enlargement of the heart does not exist.

The only cases, in which the chemical theory does not completely satisfy us, are those in which uræmic disturbances are localised and respond to circumscript cerebral lesions. We cannot recognise toxic hemiplegia nor unilateral typhoid infection, etc., and if persons under chloroform show signs of circumscribed cerebral derangements, it is because in the region attacked a less resisting spot exists, a kind of predisposing cause, be it only under the form of abnormal innervation. *Raymond's* ingenious attempt to explain the uræmic unilateral convulsions and hemiplegia has thrown some light on this subject. This experimentalist removed from one side of a rabbit the first cervical ganglion, and observed after ligature of the ureters shortly before death convulsions on the operated side, although the autopsy did not manifest any difference in the state of both hemispheres. There is no reason for rejecting *Rosenstein's* mixed theory; he unites the chemical and mechanical theories by admitting an indirect cause, blood-poisoning, and a direct one, acute uræmia of a single portion or of all portions of the brain due to toxic effect on the vaso-motor nerves. Again we must not forget *Leichtenstern's* theory; he considers inflammatory œdema of the brain and its meninges, with inconstant localisation, to be produced by resorption of scarlet fever infection without participation of retained urea, and to be the cause of scarlet fever uræmia and its rapidly changing symptoms. According to *Osthoff's* hypothesis, uræmia is due to reflex excitation of the vaso-motor centre, the termination of which is situated in the inflamed renal vessels and the tract in the splanchnic nerves.

With regard to the uræmic respiratory and digestive disturbances the *vomiting* is probably produced by a central derangement (irritation of the vomitive centre) and not by the presence of urea, etc., in the stomach; there are also other forms where an irritation of the sensitive gastric nerves of the stomach by ammonium carbonate is not improbable; the latter apparently plays a more important part in producing uræmic *diarrhœa*. *Cohnheim* attributes this diarrhœa partly to increased peristaltic produced by accumulation of urinary constituents in the blood. *M. Weiss* explains uræmic *asthma* as produced by the uræmic blood undulating the terminations of the vagus nerve, as an irritation of the motor fibres which enervate the bronchial muscles and finally as direct irritation of the respiratory centre.

Probably the disposition to inflammatory processes, which nephritic patients show, has particularly to do with uræmia in connection with hydræmia (*Traube*, *Cohnheim*).

The *mode of compensation* in uræmia resembles that in dropsy. First comes hypertrophy of the heart, which rules the excretion of the urinary constituents in such a remarkable manner. Furthermore the *sebaceous glands* can excrete solid urinary matter in a considerable quantity. The incrustation of urea on the skin of uræmic patients (uridrosis), a product of the evaporation of uric perspiration, illustrates this in a marked manner. The effect of artificial diaphoresis in uræmia is well known; still one must not forget that nephritic patients with dropsy can respond to energetic transpiration treatment with uræmia, evidently because the urinary constituents contained in the dropsical fluid are resorbed into the circulation out of places where they were harmlessly deposited (*Bartels*). Copious *vomiting*, profuse *diarrhœa* supplant in a certain degree the lessened quantity of urinary water and can thus sometimes produce a kind of natural cure. Finally, the *bronchial* and *salivary glands*, as *Fleischer* and *Thomeyer* have proved, are capable of excreting urea to a small extent (*cf.* "Treatment of Nephritis").

Diagnosis.—The diagnosis of uræmia is now-a-days much the same as *Frerichs* established thirty years ago. In cases, where kidney disease is evident, where a suitable anamnesis, where dropsy, cardiac hypertrophy, and the urine itself exist, even an inexperienced physician will nearly always recognise the nature of the above-mentioned (generally epileptiform) symptoms. It is quite another matter if indications are missing, and the patient who has appeared hale and hearty is found prostrated in coma in the street. In such a case mistake is not only pardonable, but even then, when catheterism yields urine containing albuminous casts, the presence of uræmia can be scarcely more than vaguely presumed. Not every convulsive attack, not every coma, even in notorious cases of nephritis, must necessarily be of uræmic nature; they can proceed from apoplexy, meningitis, etc., as many of the cases observed by *Rosenstein* and *Desnos* (purulent meningitis and cerebral hæmorrhage existing together with nephritis or albuminuria) teach us. In a case where the post-mortem established tubercular meningitis and genuine contracted kidney, it was impossible to decide what share each of the two processes had, as no vascular symptoms were to be found. With one exception the post-mortems of persons suffering from renal disease have shown us organic cerebral lesions (hæmorrhage, embolism, mollities cerebri) when the patients have been sent to us in a uræmic condition with unilateral or central symptoms. We agree with *Rosenstein's* opinion that paralytic symptoms in the motor regions generally speak against the presence of uræmia; we specially draw attention to its practical importance. Contrary to this author, however, we warn against too much importance being attributed to increase of temperature. In uræmia the same can be missing, in apoplexy it can be of great importance. A great number of other illnesses can be occasionally mistaken for uræmia; we mention especially epilepsy, hysteria, cerebral hæmorrhage, meningitis, typhus fever, intoxications, especially with opium and belladonna (pupils!), the diabetical coma. One has also often taken uræmia for serious local disease of the stomach and intestines, bronchial asthma, and even for stenosis of the larynx. We have made a post-mortem of a female corpse with contracted kidney, when larynx stenosis had been falsely diagnosed and tracheotomy had taken place. A similar case is described by *Runeberg*. We must strongly warn against forming a diagnosis based on uræmic symptoms alone ; a minute general examination should always precede.

The differential diagnosis of uræmia and ammoniæmia is given in the chapter on "Pyelonephritis".*

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* *Tonkin*, "Case of Uræmia following Scarlet Fever; Recovery," *Lancet*, xxiii., vol. ii., 1893.—The patient, a strong, well-built lad of sixteen years of age, came under his observation on 3rd May, 1893, and was found to be suffering from scarlet fever. The rash was well developed ; the temperature—taken in the mouth—did not exceed at any time 103 5° F., and under regular treatment the disease ran an ordinary course, the eruption fading on the 7th, and his temperature reaching the normal on the same day. Desquamation was complete before the end of the month. Now another and a more serious phase of the disorder developed. Soon after the commencement of desquamation the kidney complications presented themselves in more than usual force. Their onset was marked by suppression of the urine—total for two days, but then it gradually yielded—accompanied by a hard pulse and a slight elevation of temperature ; and when at last the secretion flowed more freely it was found on analysis and microscopical examination to contain blood, casts and a large amount of albumin. However, under the exhibition of acetate of potassium and digitalis, preceded by a brisk purge, together with hot applications to the loins, the condition of the kidneys and their secretion so much improved, and with them the patient's health, that by 2nd June he was allowed to take open-air exercise, and the perchloride of iron was prescribed. For the space of eight days at this juncture he was not under observation. On 10th June, however, at 4 P.M., medical aid was again requested, and on arrival it was found that the boy had just recovered from a convulsion which had been preceded during the morning by headache, unsteadiness of gait and some intolerance of light. He was sensible and inclined to make a jest of his condition ; subsequently, however, he denied all remembrance of what had passed. It being suspected that this fit was the first of a series, the boy was at once removed to bed, and on it being ascertained that the amount of urine passed during the previous twenty-four hours had again been scanty, hot mustard-and-linseed poultices were applied to the loins, and 60 gr. of jaborandi were given. But before the latter had time to act the right hand and facial muscles of the patient began to twitch, and almost at once he passed into a second complete convulsive condition, the interval between the two fits being thirty minutes.

The description of this attack will serve for all the others. As far as could be seen, all the muscular groups were involved in the epileptiform movements, the violence of which was so great as to necessitate restraint ; the patient foamed at the mouth, the head and neck became congested, and

The prognosis of acute uræmia is always dubious, that of chronic uræmia unfavourable.

For *treatment cf.* "Treatment of Diffuse Nephritis".

the tongue was bitten. Unfortunately, neither the exact length of time they lasted nor the variations in temperature during the attacks were noted. The convulsion over, the boy sank into a comatose condition, his face being pale and the eyes half closed; the pupils were dilated and reacted to light; the pulse was at first full, but afterwards hardened; the breathing was not markedly stertorous; the temperature was slightly sub-normal, and the extremities were cold. This state of things lasted for about twenty-five minutes, during which time hot-water bottles were applied to the feet, and then, after a short period of restless and purposeless movement, he passed into a third convulsion. For the next two hours the fits continued to succeed one another (one every thirty minutes), some differing slightly in degree from others, but maintaining, as a rule, a uniform character—severe, general, and protracted. The only difference observed was that the intervals during which the patient was comatose in the earlier stages of the series were now occupied by delirium. No amelioration being observed in his condition, it was decided, after the cessation of the seventh fit, to make a further attempt at treatment. His loins and lower limbs were enveloped in a sheet wrung out of boiling water and applied as hot as was judged possible short of scalding, and, in spite of violent opposition on his part, with the help of two men, a wooden gag, and a padded bottle, he was enabled to swallow a draught containing sixty grains of hydrate of chloral and forty grains of bromide of ammonium. The hot pack had the beneficial effect of producing free diuresis, but the convulsions still continued with unabated violence and maintained their regularity with regard to time—one every thirty minutes. In the interval that followed the fifteenth fit the bromide and chloral draught was repeated under the same difficult conditions as previously, but without any immediate effect, and on the patient completing his twentieth convulsion venesection was decided upon and immediately performed, fifteen ounces of blood being abstracted from the right median basilic vein. The blood was dark-coloured and flowed freely. The patient had two more fits before any improvement was noted, and then the attacks began to decrease in severity, and after he had had five more (making a total of twenty-eight) they ceased. The decrease in violence was by no means so marked as might have been expected, for the last fit, which, it may be remarked, was preceded by an interval of only twenty minutes, was by no means a slight one. The termination of the series might be characterised as abrupt. The patient now, after a brief interval of slightly stertorous breathing, settled down into what resembled a heavy natural sleep, which lasted for forty-six hours, and then, gradually awaking after a short period of mental confusion, entirely regained his senses. He complained of thirst, and within two hours of his return to consciousness passed urine and a large dark and solid motion. His appetite and strength steadily improved; under diuretic and tonic treatment and diet he was able to leave his bed in a week and his room two days afterwards, and on reaching the open air rapidly recovered.

The points of interest in this case seem to be the number and regularity of the fits (twenty-eight in fourteen hours) and the recovery of the patient. A specimen of the urine passed on the morning of the day that the convulsions set in was, unfortunately, not available, but an examination of the first specimen passed after his recovery showed no great deviation from the normal. The odour of the patient's breath was distinctly ammoniacal, and his skin was irritable, as evidenced by his constant scratching when his hands were not held; but there was no vomiting or diarrhœa, and no involuntary discharge of fæces during the attacks.—W. H. G.

II. SPECIAL PART.

CIRCULATORY DISTURBANCES OF THE KIDNEYS.

Kidney Disease in Asiatic Cholera.

Symptomatology.—About thirty years ago the clinical and anatomical symptoms of the cholera kidney were ably described (*Oppolzer*, *Reinhardt*, *Leubuscher* and others). Although *Koch's* wonderful discovery of the cholera-bacillus opened a new view as regards the pathogenesis, the interpretation of the pathological signification by no means belongs to recent date.

Provided the cholera patient outlives the anuric asphyxiated stage, he usually passes two or three days after the attack a small quantity of urine, poor in salts and containing albumin and casts, occasionally also epithelial cells and blood corpuscles. In favourable cases the urine is now passed freely and albumin and deposit generally disappear; at the end of the first week the kidneys generally produce normal secreta. If, on the contrary, the case becomes worse and takes a typhoid form, a renal lesion generally exists, which is clinically characterised by oliguria or even anuria. The urine is hazy, moderately albuminous and contains but little urea (*Bartels*, *Arena*) and much indican (*Wyss*), deposits plentiful sediment and owes its strong acid reaction to a considerable quantity of lactic acid (*Arena*). Microscopical observations generally manifest long hyaline and metamorphosed epithelial casts, loose degenerated (by necrotic coagulation) epithelial cells, pretty frequently also red blood corpuscles and the formed elements of a catarrh of the urinary passages. Serious derangements of the nervous system, somnolence, delirium and spasms, furthermore retarded pulse, deep respiration and uridrosis (*Hamernjk*, *Schottin*) accompany the urinary secretion. Dropsy never appears. Death generally occurs after two or three days under general paralytic symptoms. Recovery is rare, and in cases of more than five days' anuria, impossible. Transitions into lingering inflammation of the kidneys do not take place (*Bartels*).

Pathological Anatomy.—The post-mortem generally shows tolerably enlarged kidneys, with broad, dull, greyish-red or yellowish-grey cortex, which is often slightly speckled, and bluish-red medulla. The microscope usually manifests, according to the latest experiments (*Ceci*, *Klebs*, *Strauss*, *Roux*, *Nocard*, *Thuillier* and others), a decided coagulative necrosis (*cf.* "Renal Infarct") of the epithelium, especially of the tubuli uriniferi, and no particular fatty degeneration; still *L. Meyer* seems to have observed such pronounced forms of fatty degeneration that certain parts of the cortical region had the appearance of a creamy pulp. Small hæmorrhagic centres are often found simultaneously in different places. The process is generally cured by epithelial regeneration (*Buhl*). The anatomical integrity of the glomerular loops, already asserted by *Virchow* and *Güterbock*, has been confirmed by all observers. The interstitial tissue appears equally intact (*Kelsch*). The cholera kidney is often accompanied by a catarrh of the pelvis and calices. Nearly all experimentalists of the present day agree that *Koch's* comma bacillus is not to be met with in the kidneys; only a few (especially *Doyen*) declare that they have noticed an invasion of micro-organisms.

Although the most competent authorities consider the cholera kidney to be identical with Morbus Brightii, an inflammatory process must be strictly negated. The principal cause must rather be sought for in the increased effusions into the intestines, which produce circulatory derangements. Thus we have to do with an *ischæmia* of the kidneys in consequence of excessive inspissation and protraction of the blood current. This opinion, which *L. Meyer*, *Buhl*, and above all *Griesinger* uphold, and which has been lately most strongly defended by *Bartels* and *Cohnheim*, is not without experimental corroboration. We meet with analogous alterations, stoppage of the urinary secretion, with transudation of albumin and casts, epithelial lesions, with artificial hindrance of the circulation through obstruction or temporary ligature of the renal arteries (*Overbeck*, *Hermann*, *v. Platen*, *Litten* and others). To what extent an injury of the renal epithelium, produced by ptomaines formed by the comma bacillus, concurs (alkaloids, *Nicati* and *Rietsch* and others), still remains to be proved. The comma bacilli themselves play no part. The above-mentioned serious complex of symptoms resembling typhus is nothing else than uræmia; *Oppolzer*, *Hamernjk*, *Frerichs* have already recognised it as such. *C. Schmidt*, *Buhl*, *Voit* and others found the whole organism overloaded with urea. Anyhow, the resemblance of the typhoid condition with typical uræmia is certainly, as *Rosenstein* rightly remarks, very slight, and in many cases *Griesinger's* irritative fever theory (too sudden calefaction of the algid patient) is not to be underrated.

Renal disease in cholera shows various transitional forms towards albuminuria, partly in consequence of intestinal catarrh, partly in consequence of great loss of blood, especially hæmatemesis (*Eichhorst*), also in the course of serious anæmic and cachectic conditions, perhaps also towards febrile albuminuria in the course of acute infectious diseases, in as far as an effect of soluble excretory products of micro-organisms comes into consideration (*cf.* the part on "Albuminuria" and "Acute Nephritis").

Prognosis.—The quantity of albumin does not afford a conclusion regarding prognosis (*Bartels*). The presence of numerous casts at the beginning is considered a favourable symptom (*Wyss*). We have already mentioned that the patient is even lost if urinary excretion does not take place on the sixth day.

Treatment.—The treatment must be in accordance with the primary disease. Administration of various analeptica and of great quantities of fluid stands in the foreground, the latter especially in the form of *Samuel-Cantani's* sub-cutaneous injections of alkaline saline solutions and plentiful incorporations of warm tannin, and similar solutions into the intestines ("hypodermoklysis, enteroklysis"). Although an even tolerably certain effect of this method cannot be always expected, yet it occasionally prevents renal affection from taking place, and often lessens the danger.

RENAL AFFECTIONS DURING PREGNANCY.

The following description limits itself to kidney disease during pregnancy, which under certain circumstances can form the basis of the much-feared epileptic form of attacks known as *eclampsia gravidarum*, *parturientium*, *puerperarum*. The malady is fortunately not very common. Those cases, however, where renal disease already existed before pregnancy, or where a genuine nephritis formed itself during the same in consequence of different causes, do not belong to this category; the same may be said of albuminuria as sequel to labour. Differentiation of these cases is by no means easy.

Lever was the first who, forty years ago, brought eclampsia into connection with albuminuria, and made the pressure of the pregnant uterus upon the renal vessels responsible for it. Ten years later *Frerichs* declared eclampsia to be a uræmic complex of symptoms.

Symptomatology.—Puerperal nephritis most frequently occurs with young primiparæ in the last months of pregnancy. Twin pregnancy is a pre-disposing moment. Seldom, however, does a marked manifestation allow of the precise distinction of the commencement of the illness. A small quantity of albuminous urine is passed. The other initial symptoms, such as strangury, pains in the renal region, sickness, vomiting, œdema of the feet, are often met with in the last months of pregnancy, while urine and kidneys are quite normal. It is different if *dropsy*, which generally precedes albuminuria (*Lehfeldt*), attacks the lower extremities *in toto*, the genitals, the upper half of the trunk and the face. In other cases dropsy and all other symptoms do not appear, and only by testing the urine can we discover the disease. The urine is remarkable for decrease of quantity, increase of specific gravity, light colour; the quantity of albumin is often very considerable, according to *Bartels* and *Spiegelberg* several per cent. (in one case we saw the urine become totally coagulated on ebullition), but is, however, in no proportional relation to the dropsy. Decided hæmorrhagic character is an exception. The deposit is minimal and contains delicate and firm hyaline, epithelial and granular casts, loosened and fatty renal epithelial cells, fat granules (*Leyden*), sometimes lymph corpuscles, and now and then red blood corpuscles and hæmatoidin crystals (*Hiller*). In a case which came under our observation, and which was rapidly cured, the ample urine deposited a copious sediment containing almost exclusively broad metamorphosed and fatty granular casts.

Out of the number of pregnant women suffering from albuminuria about a quarter are attacked by eclampsia (*Rosenstein*). It generally appears towards the end of pregnancy, *during* birth, less often before the beginning of labour or during childbed (*Braun*, *Wieger*). Out of every 500 births one case of eclampsia is said to occur. The convulsions generally occur suddenly without precursory symptoms, such as depression, somnolence, headache, vertigo, vomiting and slight spasms. The paroxysms are remarkable for violence and frequency; often thirty follow consecutively. The attack itself resembles an epileptic fit; sometimes the tonus is predominant, sometimes the clonus. Cyanosis and stertor accompany the paroxysms, the patient foams at the mouth and bites her tongue; increase of temperature is in general parallel with the intensity of the attack. The attack lasts a few minutes, or even only a few seconds. After the first attack consciousness returns, and remains later on during the intervals of the coma. Maniacal excitement often interrupts the sad scene. Sometimes the patient awakes totally blind; having known nothing of the birth which has taken place. Death occurs in thirty per cent. of the cases (*Rosenstein*, *Dohrn*, *Löhlein*). Death takes place sometimes when the attack is at its climax from pulmonary œdema or apoplexy. When the course of the attack is favourable the patient generally recovers quickly with plentiful diuresis; already a few hours after the last attack the urine can become normal. The more acute the course, the easier is the complete recovery. Besides puerperal processes, serious after-effects, secondary inflammation of internal organs are always to be feared. With a multipara we have seen a spontaneous miscarriage followed by several attacks of eclampsia. The urine remained albuminous, all pathological constituents disappeared with the recovery from severe pneumonia, which followed the last attack.

Morbid anatomy.—Former exponents have already remarked that puerperal nephritis is able to transform itself into genuine chronic Morbus Brightii. The recent observations of *Leyden*, *Weinbaum* and *Herrgott* have confirmed this observation. *Flaischlen*, however, insists that this occurrence is very uncommon. Albuminuria often lasts weeks and even months. The children are even more seriously endangered than the mother; one half of them succumbs. On dissection a uræmic soft and pale yellow organ will be found with moderately tumefied cortex and more or less hyperæmic pulp. Microscopical examination usually shows tolerably intact glomeruli and connective tissue; the epithelial cells of the tubuli uriniferi, especially of those of the cortex, have undergone various stages of adipose degeneration; the lumina of the tubuli contain the above-mentioned casts and are sometimes filled with blood corpuscles. In one case *Leyden* observed the convoluted glomeruli replete with masses of fat globules, an evidence which *Virchow* corroborates for certain cases in somewhat modified form, in stating that it is no primary disease of the glomeruli, but the sequel of a genuine fat embolism, and that the glandular cells of the kidney are not the seat of a degenerative fatty infiltration. *Leyden*, however, gives this explanation because the reagents which extract fat leave them intact (*Hiller*). *Virchow* does not deny a certain degree of adipose degeneration, but, on the other hand, does not believe that the kidney without epithelial lesions resembles lipomatosis hepatis. The description which we have given can be modified with regard to disturbances which proceed direct from pregnancy or are connected with mycotic embolisms proceeding from the endocardium, or finally from a phenic intoxication. Complications with catarrh of the urinary organs and also pyelonephritis (*Leyden*) are often met with; *Jürgens* declares to have constantly seen such in post-mortems with hæmorrhage of the liver and lungs of bacillary origin. Yellow atrophy of the liver is also met with (*Stumpf*). Cerebral anæmia and œdema are no uncommon accessories (*Schauta*). In such cases where fat embolisms are found in the glomeruli and intertubular capillaries of the kidney and in the pulmonary vessels they are not necessarily apparent in the cerebral vessels (*Virchow*). According to *Wiedow* concomitant disease of the placenta (white infarctus) may be considered as a consequence and not the cause of renal derangement. In cases of eclampsia without albuminuria the kidneys are generally intact; in other cases chronic Morbus Brightii surprises us, which remains latent *intra vitam*.

Pathogenesis.—The nature of puerperal nephritis is still very inexplicable. The theories of its origin are numerous; it is impossible to decide entirely in favour of one or the other. There are probably various forms, each with different origin. In a number of cases the attacks are decidedly of a uræmic character—recently *Herman* noticed during an attack a quite considerable decrease of urea in the urine, which later on contained enormous quantities (*cf.* chapter on "Uræmia")—without the diagnosis of genuine nephritis being justifiable. It should rather be considered as a special form combined with anæmia and adipose degeneration of the cortical substance without permeability of the glomeruli in favour of albumin. But where are the etiological moments? The accused changes of the sanguinary crasis in pregnancy, hydræmia, can only be considered as more remote cause, not as original basis; it must be remembered that hydræmia is no constant symptom, and that often the healthiest women are attacked by puerperal nephritis; again hydræmia itself does not cause albuminuria. Another opinion finds the causal moment in the topography of the pregnant uterus with regard to the urinary apparatus. Compression of the *renal veins* producing consecutive renal engorgement is unlikely; the engorged kidney is cyanotic and hard, the puerperal kidney pale and soft, and the form of the disease does not assume the clinical symptoms characteristic of venous hyperæmia. Above all the topographical anatomy of the kidneys speaks against the probability of pressure on the renal veins (*Bartels*, *Schröder*, *Ingerslev*); *Litzmann* has even observed the malady in cases when physconia prevailed. The assumption that the pregnant uterus presses upon the *renal arteries* is on topographical grounds equally inconsistent. At first sight the theory so warmly advocated by *Halbertsma*, that the principal forms of eclampsia are due to pressure or traction exercised by the pregnant uterus upon the *ureters*, appears more plausible; retentio urinæ ensues, which in its turn causes the uræmic attack. In consequence of the low renal pressure obstruction of the bent and distended ureters is possible, hence predominance of eclampsia in primiparis and twin pregnancy, as well as the frequent attacks of convulsions during labour, which are conducive to urinary obstruction and the not uncommon complication with pyelitis. *Halbertsma's* theory claims certain probability, but is wanting in decided confirmation. In a small number of cases observed by *Löhlein* distention of the ureters was found post mortem. Experimental compressure and ligature of the ureters produced albuminuria together with oliguria (*Posner*), dropsy and convulsions (*Popoff*), fatty degeneration of the renal cortical epithelium together with formation of casts (*Aufrecht*). Œdema of the kidneys, a further postulate of the experiments (*Senator*), was also produced. Nevertheless, fundamental importance cannot be attached to this theory, as other tumours, exercising much greater influence on the ureters than the pregnant uterus, do not produce eclampsia, and as animals can bear obstruction of the ureters for three days and more without presenting uræmic symptoms. Finally, we will mention *Cohnheim* and *Spiegelberg's* theory, a theory which is now nearly abandoned and according to which *spasm of the renal arteries* impedes the blood supply to the glomeruli and thus causes retention of urinary constituents in the blood. It is true this hypothesis would explain why "sound" kidneys are to be found in particularly rapid cases, and why the symptoms disappear so quickly; but, nevertheless, these considerations in no way justify the idea of a vascular spasm of several days' duration. Recently a theory, much resembling that of retention, has come into the foreground, *i.e.*, the idea that eclampsia can be caused by a *specific poison* of pregnancy. We must assume that this theory refers less to an intoxication with the intermediate products of decomposition of an abnormal circulation (*Stumpf*, *Galbain*) than to toxic secretion of specific *micro-organisms*, to the successful invasion of which pregnancy predisposes. *Doléris* and *Poney* have found in the urine of persons suffering from eclampsia microbes, which when injected into animals cause death from albuminuria and convulsions. *Doléris* and *Butte* bled an eclamptic patient and found in the ethereal extract of the blood a crystalline toxic substance, which first had an exciting, later on a paralysing, effect. Urea was not increased. *Virchow* reports the discovery of mycotic endocarditis in a case of eclampsia.

The relation between eclampsia and *fat embolism* has been proved by *Virchow*. This co-relation is by no means constant and is not remarkable, since *Busch* has observed that particles of marrow brought into the circulation have a predilection to settle themselves in the kidneys. *Virchow* has demonstrated that it is the obstruction of the pulmonary capillaries which has the lethal effect. As injuries and contusions of the subcutaneous connective tissue in pregnancy are numerous, one is not at a loss to discover the origin of fat embolisms; perhaps the liver furnishes also embolising material (*Zenker*).

Schröder, *Ingerslev* and above all *Rosenstein* advocate a theory independent of blood poisoning or of obstruction of pulmonary circulation. The same corresponds with *Traube's* hypothesis of uræmia. They declare that eclampsia is dependent on modifications of *cerebral circulation* combined with œdema and *acute anæmia* of the brain. *Rosenstein* considers the simultaneous occurrence of renal disease only to be an auxiliary and not causal moment of eclampsia, as the kidneys can remain quite intact and eclampsia can appear without albuminuria; furthermore as pregnant women suffering from chronic *Bright's* disease do not necessarily have convulsions, as eclampsia is in direct connection with labour itself, and finally as the brain shows certain anatomical indications. *Ingerslev* declares the concomitant renal disease and *vaso-motor reflex neurosis* as essential cause. Diffusion of the blood serum and increase of pressure in the aortic system in consequence of labour are supposed to be the cause of cerebral anæmia. Independent of the restrictions already given, experience teaches that eclampsia only exceptionally appears in cases where albuminuria is not present, and at a period when labour, after-pains and hydræmia do not exist.

Finally, we draw attention to *Osthoff's* theory, already mentioned, and remark that according to *Spiegelberg* a part of the attacks of eclampsia are nothing further than *acute epilepsy*, of which the epileptogenous zona is limited to the ischiatic region. *Lantos* has recently regarded eclampsia as "epilepsia acuta peripherica". According to him the pressure of the pregnant uterus is an excitative moment, which has a reflex action on the medulla oblongata. He also attributes the concomitant albuminuria as subsequent to the reflex irritation of the renal vessels.

Individual predisposition naturally influences all these theories.

Diagnosis.—Puerperal eclampsia can be mistaken for genuine epilepsy and hysterical convulsions, especially as albuminuria is no diagnostical symptom when found in pregnancy. The anamnesis generally helps to decide the point, and the absence of deep coma speaks for hystero-epilepsy. One must not forget that serious organic cerebral diseases (meningitis, hæmorrhage, tumours) can occur during pregnancy and labour. Deficient knowledge of the case can cause great difficulty in distinguishing the puerperal nephritis from chronic nephritis. The surest sign of chronic nephritis is doubtless hypertrophy of the heart and characteristic hard pulse (*Flaischlen*). Still we have seen a case unexpectedly cured in a few days in spite of these symptoms, and in spite of low specific gravity of the urine.

Prognosis.—The *prognosis* is, in spite of some favourable statistics, dubious. When once convulsions appear, the life of the mother, as well as that of the child, is always in jeopardy; the fourth part die. The nearer the attacks of eclampsia take place after delivery, so much the more favourable is the prognosis (*Lohlein*); it is an equally good symptom when the attacks end soon after delivery, and the patient recovers consciousness in a day at the latest. *Schröder* attaches great importance to the state of the pulse; slight tension and fulness render the prognosis almost absolutely bad. Puerperal nephritis has of itself a favourable prognosis, in as far as transition to chronic *Bright's* disease is an exception.

Treatment.—Dropsy gives an indication for diaphoresis. The most harmless form is *Liebermeister's* method (*cf.* "Treatment of Nephritis") (*Bartels*). It never occasions a miscarriage nor attacks of eclampsia. *Breus* has had out of twenty-four cases twenty-two cures. Nevertheless, precaution is necessary in cases of debility of the heart. We do not recommend pilocarpine so much prescribed by *Cantilena*, for it produces hæmorrhage (*Hiller*) and attacks of suffocation (*Sänger*), also reflux of saliva during the coma. True to his theory, *Osthoff* warns against the administration of digitalis, as the irritation of the vaso-motor centres could, in consequence of augmented pressure, irritate other centres. With weak hurried pulse this remedy and camphor is, nevertheless, according to our opinion, of the greatest service. A few rather unfavourable experiences have taught us to avoid *drastics*. For the attack itself one administers *narcotics*, even chloroform narcosis (*Schröder*), hydrate of chloral as enema and morphia. *Veit* recommends the latter to be administered in very large doses (up to 0·04 subcutaneously), combined with transpiration baths, and proves this treatment with exceedingly favourable statistics (two deaths out of sixty cases). (For bleeding and other methods *cf.* "Treatment of Uræmia".) *Chambert* only allows of venesection in cases of plethora. In France an absolute milk diet is considered very beneficial. With regard to accelerated termination and artificial premature delivery, which is sometimes necessary in serious cases, and which *Pooley* strongly advocates when impairment of vision takes place, one finds instructions in the manuals on midwifery. *Schröder* pleads for artificial abortion under the plea that pregnancy tends to turn the puerperal state into chronic nephritis.

ENGORGED KIDNEY.

Etiology and pathogenesis.—This disorder, subsequent to serious circulatory derangement, does not in general appear as an independent organic affection, but as a *symptom* of various primary disturbances. According to the mode of development two principal forms can be recognised :—

(1) *Local serious engorgements subsequent to contraction or obstruction of the renal veins or of the vena cava inferior below the inosculation of the renal veins.*—Pressure by various tumours and consecutive thrombosis plays an important part; the latter, however, can also develop itself independently as marantic form in cases of serious cachexia, in cases of cholera nostras in children, especially in infants (*Beckmann*, *Pollack*, *Schwartze*). The cases belonging to this category are less frequent than those produced by general engorgement.

(2) *General engorgement of the venous system, subsequent to derangements in the pulmonic circulation.*—General arterial anæmia exists at the same time. Valvular disease, especially of the venous ostia, when insufficiently compensated, is the most frequent cause of renal engorgements. Aortic derangements generally cause symptoms of nephritis and not of engorgement. Diseases of the textures, such as inflammation, arterio-sclerosis, inflammatory degeneration, and, above all, dilatation of the cardiac cavities of the heart and atrophy of the myocardium, can in their turn produce renal engorgement. Besides these organic heart diseases pleuro-pulmonary affections can become an obstacle to general circulation, such as emphysema, contraction, pleuritic

exudation. On the contrary, it is an uncommon thing to find engorged kidney in cases of phthisis, a fact to be explained by the concomitant poverty of blood. Chronic engorged kidney is to be distinguished from the acute form, a type which, strange to say, is seldom mentioned in text-books. We have met with the acute form in the post-mortems of such individuals who died from suffocation. Suffocation from hanging, drowning, transplacement of the large bronchus (croup, corpus alienum) plays a prominent part, also acute lung and heart affections (pneumonia, pericarditis), finally, certain poisons of suffocative effect (prussic acid, etc.). *Schreiber* has succeeded in rendering the urine of healthy individuals albuminous through simple compression of the thorax, and finds the reason in a restriction of the respiratory change of the volume of the lungs and in the rapid propagation of the engorgement of the kidneys.

Pathological anatomy.—We will not enumerate all the different disorders capable of producing thrombosis by compression of the vena cava or renal veins. The forms of thrombosis are very various (total obstruction, parietal thrombus, perforated in the centre, etc.). It would seem that the left renal vein is more frequently affected. Sometimes the kidney becomes the seat of an intense hyperæmia—we have seen in a case of an infant who died of cholera a dark brownish-violet succulent kidney, on section a tarry liquid oozed out—or does not differ much from the normal organ because an adequate collateral circulation forms itself.

Engorged kidney, as a sequel to *general* venous obstruction, so frequently found post mortem in persons who have suffered from heart-disease and emphysema, appears seldom more than moderately enlarged, hard, superabounding in dark blood, especially in the region of the veins and surrounding capillaries. The medullary substance, especially towards the basis, appears darker than the cortex, the surface of which is often remarkable for the designs called *Verheyn's* stars, while the papillary portion is generally of a paler colour. This is the so-called *cyanotic induration* (*Klebs*). Provided the processes are of not too long duration, microscopical examination generally manifests unchanged epithelium, broad fibrous structure in the medulla (*Paricelli*) and somewhat extended interstices. In very chronic cases the organ is generally diminished in volume; its cortex somewhat atrophied, and the capsule can only be stripped off with difficulty from the nodular surface, and portions of the tissue come away with it. In such cases we find the capsules of *Malpighi* thickened, the glomeruli slightly shrunk, the epithelial cells in a granulated or fatty degenerated condition and cellular infiltration of the tissue. Finally, combination with genuine nephritis or *contracted* kidney is no seldom occurrence; we use the term combination because, according to our opinion, we have not to do with simple sequels of engorgement in the sense of *Freylinck's* experiments, but as *Rosenstein* remarks, with co-efficients of the same primary disorder; probably not of rheumatism alone, but of endocarditis in the word's widest meaning. We have met with the large atrophied, granular, engorged kidney—with the great number of heart affections in the hospital Friedrichshain, it is quite usual—in cases of valvular affections of the heart, the non-polyarthritic origin of which can be regarded as proved, as well as in ordinary rheumatic endocardiac affections and their sequels. Nevertheless, we have only exceptionally seen engorged kidney transform itself into the typical small contracted kidney, while we have observed all possible transition towards cyanotic induration, as nearly twenty-five per cent. of all cases of engorged kidney. Such cases present a combination of symptoms of engorgement and of *Bright's* disease without such decided lesions of the secretory portions as found in genuine *Bright's* disease. Therefore we classify these cases—their clinical symptoms are also in accordance—as engorged kidney and not *Bright's* disease, although it is sometimes very difficult to distinguish during life, and even post mortem, especially when a complication with arterio-sclerosis exists.

The acute engorged kidney has more or less pronounced venous hyperæmia, the line of demarcation being of a dark, bluish-red colour. Other changes are not to be found.

One must not forget that acute as well as chronic engorgement can appear as an accessory to *Bright's* disease. Then mixed forms appear, which can resemble the above-mentioned, and are often enough mistaken for them.

Symptomatology.—The symptomatology is more or less congruent with the primary disease. The condition of urine is of most significant importance.

A remarkable fact is that in a number of cases of the already mentioned *first* category, even with total thrombosis of both renal veins, deviation of the general urinary character is not to be found. They are those kinds in which the thrombosis is occasionally found at the post-mortem, and the kidney itself presents no change of structure. Such occurrences appear all the more strange as the experiments with animals show positive results.

Apart from those consequences of a ligature of the renal veins, which leads to enormous hyperæmia of all renal vessels with copious extravasations of blood which cause the organ to excrete lymph instead of urine (*Cohnheim*), or to excrete nothing at all (*Senator*), and which finally cause atrophy, *Perls* and *Weissgerber* have produced by obstruction of the renal veins excretion of albumin, of blood and of casts. Quite short venous obstruction recently effected by *Senator* caused decided albuminuria.

Accordingly, we see that if in human pathology the effect of renal obstruction on the excretion of urine is not coincident, this can only be due to the *slow* development of the thrombosis, together with a considerable decrease of arterial pressure in consequence of cachexia. Thanks to this slowness on the one side, and to the decrease of arterial pressure on the other, the whole blood supply brought by the arteries is carried by the collateral tract of the capsule veins (*Cohnheim*). There are cases, however, where the morbid phenomena found in human beings quite resemble those found in animals. *Bartels* has observed a *robust* man with a voluminous thrombosis in the vena cava inferior. He remarked albuminuria and hæmaturia with considerable increase of urine, the latter doubtless due to high pressure in the glomeruli. *Oppolzer* and *Nottin* likewise found in cases of thrombosis of the renal veins abundant quantity of albumin and blood in the urine. *Pollack* and we ourselves have found the same in cases of cholera nostras.

In those cases where engorged kidney appears as a symptom of *general* venous obstruction in consequence of organic debility of the heart, we usually meet with the following clinical type: the patient has noticed for a considerable time a decrease and a dark colouring of the urine and is alarmed by a dropsical swelling of the feet. If one examines the urine at this period one finds a daily quantity far below the normal, often reduced to the half, its specific gravity is increased and generally reaches 1030. The brownish-red, highly acid excreta usually deposits, after becoming cold, a great quantity of urates and also crystallised uric acid; its quantity can amount to one-twentieth part of urea contained (*Bartels*). The absolute quantity of the latter is nearly normal when the functions of the renal epithelium are but little disturbed; when, however, serious digestive derangements and cachexia concur, the specific gravity is relatively and even absolutely decreased. Dropsy having once put in an appearance, albuminuria is most frequently met with, not constantly, however, as *Bartels* declares. The quantity of albumin is, as a rule, very small, containing one or a fractional part of a gramme per mille, but we have found in even genuine cases, contrary to the opinions of some exponents, a relatively considerable quantity (more than 0·5 per cent.). The microscopical examination of the scanty sediment usually confines itself, apart from the urates and the formed elements of the nubecula, to few hyaline casts (compare fig. 1), and occasionally to some red blood corpuscles.

Regarding oliguria contradictory opinions exist. Although the but slight injection of the vascular loops post mortem seems to speak for the theory, that oliguria is produced by decrease of blood-pressure in the glomeruli (*Runeberg*), the argument might be raised that venous obstruction extends itself to the glomeruli and establishes an increase of pressure (*Heidenhain*). The explanation given by *Senator* and *Cohnheim* is *difference* of pressure inside the glomeruli and the tubuli uriniferi. The extension of the engorged blood-vessels, especially in the medullary substance, leads to compression of the tubuli uriniferi, this leads to urinary engorgement (*Ludwig*); the contra-pressure more than compensates for the increase of tension in the glomeruli. Very likely the retardation of the blood current in the vascular loops plays also a part (*Heidenhain*) and can at the same time be made responsible for the appearance of albuminuria. Indeed, as *J. Paneth* has recently proved in dogs by experimental ligature of the vena cava, the mechanical part of the compression of the tubuli uriniferi must be excluded, and the oliguria be considered solely due to injury of the secretory function of the kidney produced by venous obstruction without any participation of arterial pressure, as diuretics (saltpetre) immediately produced abundant secretion of urine. According to *Senator's* experiments, dissolved albumin and blood corpuscles enter, as already mentioned, into the urine in the form of congested lymph direct out of the abnormally distended capillaries of the medullary substance. The results of the above-mentioned experiments in animals by which albuminuria and transition of red blood corpuscles are produced, cannot, as *Senator* remarks, be applied to the human engorged kidney, as immense circulatory derangements are caused by the venous obstruction with *unchanged arterial circulation.* By the latest experiments of *Munk* and *Senator* it appears that the quantity of urea, but not that of chloride of sodium, decreases proportionally in the urine under the influence of venous obstruction, and we must conclude with *Paneth* that oliguria is closely connected with a lesion of the renal epithelium, and, as already remarked, to a transudation through the walls of the tubuli uriniferi.

The *dropsy* which accompanies the cyanotic induration of the kidneys can scarcely deny being originated from engorgement. It ascends, develops itself the most in the region of the inferior vena cava, without, however, being confined to this part. The face and upper extremities generally remain intact, and dropsy never commences in these parts, as is so often the case with nephritis. Uræmia is absent in cases where nephritic complication does not exist.

The *course* of the engorged kidney depends naturally on the nature of the primary disease. Dropsy can exist a long time and then accompany the patient to the grave. This is the general rule for all cases where degeneration of the cardiac muscle or combination with severe nephritis does not hinder compensation; retrogression of intense symptoms of venous obstruction, be it that compensation occurs spontaneously, be it that it is artificially produced, must be considered as a remarkable characteristic. Even in the most severe and apparently hopeless cases the threatening symptoms disappear sometimes rapidly under plentiful diuresis and cessation of albuminuria. But relapses are generally lurking near at hand. The above-mentioned mixed forms of engorged and nephritic contracted kidney generally have, according to our experience, urine which has much the same character as that already described, only that oliguria, high specific gravity and dark colour are less apparent; one observes, on the other hand, a greater quantity of albumin and a number of granular and even waxy casts. Where the character of the typical contracted kidney is more marked, the urine takes the quality of this form of illness. We only remark that the quantity of urine seems to us considerably diminished, even when hypertrophy of the left ventricle was evident. Without exception the specific gravity is always remarkably low, even in combination with considerable oliguria.

Diagnosis.—Most authorities consider it impossible that chronic nephritis and engorged kidney can be mistaken. They assert that the concentration of urine, the large deposit of urates and above all the quantity of albumin is a differential safeguard. Those who consider a small amount of albumin as a pathogenic symptom of engorged kidney will certainly diagnose more cases of chronic nephritis than are found post mortem. We have often had the opportunity of observing cases with secretion of albuminous urine and general dropsy lasting for weeks, in which the pathological-anatomical examination of the kidneys showed only simple congestive disturbances (especially in consequence of myocarditis). In by far the most cases general cyanosis, the existence of a causal heart or lung disease, the mode of development and localisation of dropsy, as well as a small quantity of hyaline casts, prevent errors. In the mixed forms, already mentioned, diagnosis can become almost impossible, even although quicker progress, hæmorrhagic urine with renal casts, shows the complication in an unmistakable manner. We ascribe very considerable diagnostic importance to a *continuous* low specific gravity (under 1012) *in spite of oliguria* and general congestive symptoms; wherever we have met with them, without inanition or considerably developed cachexia concurring, we have usually found the complication with genuine contracted kidney.

Prognosis.—The prediction of renal congestion is naturally dependent on the original cause. As irreparable organic lesions generally exist, the prognosis is founded on the degree of compensation. We have seen that even intense dropsy and almost complete anuria is not necessarily fatal, and that a temporary cure is possible.

Treatment.—The treatment depends on the original disease and on the whole condition of the patient. The principal aim is to remove urinary obstruction and dropsy; the sovereign remedy, taking well into consideration the part the heart plays, is *digitalis*. In the second place *coffein* and *alkaloids*, diuretics which, according to *Grützner*, *Lépine*, *Munk*, etc., have an action "a fronte," *i.e.*, a peripheral one, on the kidneys through increase of the celerity of the circulation, and through irritation of the glandular cells. Amongst these remedies we reckon *calomel*, the paroxysmal diuretic action of which has been recently affirmed by *Jendrassik*. Our clinical observations and *Rosenstein's* experiments prove that this drug acts on the renal epithelium and not on the heart. Finally, *strophanthus* (*Fraser*) (the effect of which is not yet fully known) deserves recommendation. We cannot possibly enter here into detailed indications (which, besides, are not yet satisfactorily explained), but can recommend, however, from our experience, digitalis together with acetate of potassium and strophanthus in such cases where digitalis alone, even in large and frequent doses, remains without effect. Coffein alone, likewise strophanthus, often proves a failure, but the latter can produce in certain cases (in our experience, especially in fresh ones and such as are not complicated with stenosis of the mitral valve) abundant diuresis without discomfort, as the additions to the at first somewhat optimistically coloured reports (*Pius* and others) have proved (*Langgaard*, *Haas*, *Zerner* and *Löw*, *Hochhaus*, *A. Fränkel*, *P. Guttmann*, *Fürbringer* and others). We recommend the two last-named remedies, not to be administered alone as a substitute for digitalis, but in combination with it, for we have remarked in hopeless cases, where one remedy alone was useless, that through the mixture, enormous quantities of urine (six litres and more *per diem*) were secreted with quick disappearance of the intense dropsy, and that apparently dying patients have rallied for weeks and even months. Absolute inefficiency of these three drugs is, according to our experience, a true sign that the end is not far off.*

In certain cases we agree with *Meyjes*, *Cohn*, *Weinstein*, *Orioli*, *Stintzing*, *Bieganski*, *Nothnagel*, *Pal*, *Erb*, *Biro*, *Buschujew* and others that calomel (as altogether acute mercurialisation) is an effective, but at the same time uncertain, diureticum, which we only dare to recommend as *ultimum refugium*, but warn against in cases of complicated nephritis. With the usual doses three times daily, 0·2 grm., serious stomatitis and enteritis generally occur, in spite of all precautions, and many patients of this category succumb all the quicker without increased diuresis, which is mostly hindered by parenchymatous nephritis. The effect is nearly always ephemeral, as even the greatest advocates of this remedy must own. Nevertheless, we must allow that a certain number of cases are benefited by the use of it. On the other hand, those cases in which *adonis vernalis*, *convallaria* and

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* This experience we can also confirm with several illustrative cases, cases where extreme dropsy surrendered to the joint action of these three drugs, and where death occurred in spite of all other medication when they became inefficacious.—W. H. G.

spartein, recently so much recommended, exercise a beneficial anti-hydropic effect, we as well as *Hiller*, *Leubuscher*, *Stössel* and *Eichhorst* must consider as decided exceptions. Latterly we have given up administering these remedies, likewise *pilocarpine*, because their effect in heart disease seems deleterious.*

We agree with *Bartels* that revulsion towards the intestines is for any length of time harmful, and in cases of cardiac debility decidedly noxious, because the digestive disturbances and loss of albumin *per via* intestines which frequently ensue, can endanger the already weakened heart. In cases where the heart is very weak one should not be too sparing with stimulants (wine, camphor) (*cf.* "Treatment of Diffuse Nephritis ").

In many cases this treatment will not suffice ; then diaphoresis must be employed ; details of which are to be found in the chapter on the " Treatment of Dropsy in Nephritis ".

If none of these methods, not even in combination, are of effect, and if symptoms caused by dropsy are on the point of endangering life, *then*, but *only then*, the fluid must be surgically removed, of course always with every antiseptic precaution. For description of the procedures employed *cf.* " Diffuse Nephritis ".

The dietetic mechanical treatment of the circulatory disturbances producing engorged kidney, as especially founded by *Oertel*, we may omit here. In cases where the body is filled with dropsical fluid, and the patient requires to be nursed in bed, all meditation over a method of prophylactic value is most superfluous. The discussions of *Oertel* and *Lichtheim* give evidence, how quite lately exponents vary in their opinion regarding the importance of the diminution of the quantity of fluids partaken of, regarding terrain cures and other cardiac gymnastics in circulatory disturbances.

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* Sugar of milk, so strongly recommended by *Sée* as diuretic, we have never seen to be efficacious; on the other hand, I have witnessed good results from the administration of diuretine, in cases of oliguria and dropsy of cardiac, or especially renal, origins. As its action is—doubtless—more or less limited to the renal parenchyma, without affecting the central nervous system, I cannot agree with *Caussade*, who considers the action of the drug as merely ephemeral, having seen cases of nephritis where for a length of time the quantity of secreted urine has been increased four- to six-fold, and never less than one-half. A great mistake is made in administering too small doses ; the failure in result is then imputed to inefficacy of the drug. *Per diem* 4-6-7 grm. should be given in watery solution without addition of any acid or syrup, as otherwise the theobromine is precipitated ; likewise ordination as powder must be avoided, the carbonic acid in the atmosphere disassociating the theobromine from its natro-salicylicate and rendering the former insoluble. ℞ Diuretini, 5,0·7,0 ; aqua menth. pip., aqua dest., āā, 75,0. Headache, nausea and vomiting are exceptions.—W. H. G.

Hæmorrhagic Renal Infarct.

Renal infarct is not of great interest from a clinical point of view. It results, as *Virchow* was the first to show, from the obstruction of a branch of the renal artery by a non-infectious embolus, such as principally appears in the course of left endocarditis (see part on "Nephritis"). This embolism shows itself particularly in valvular derangements. One seldom meets with it in the course of arterio-sclerosis and renal disturbances of traumatic origin. The detailed description of embolism belongs to general pathology; we confine ourselves here to the citation of the most important facts.

Pathological Anatomy.—Renal infarct is falsely named hæmorrhagic. What *Beckmann* hinted years ago against *Rokitansky's* theory of the discoloration of the so-called hæmorrhagic deposit (fibrinous wedge) has been confirmed after careful experimental observations by *Litten* and *Talma.* Genuine hæmorrhagic renal infarct is *uncommon*, the majority is *white* infarct, which is not caused by a discoloration, but by *anæmic* necrosis, which on its part is unconnected with hæmorrhage. Such infarcts do not present themselves at first as blackish-red and later on as light-coloured centres, as described in recent manuals; they are colourless from the beginning, corresponding to the ramification of the affected vessel, wedge-shaped or conical with the apex in central direction. They are white, yellow or greyish; this colour explains the fact that former observers have called them, when particularly small, puriform deposits. Finally, the infarcts are dry and have on the periphery a red line of demarcation.

Microscopical examination shows that here, as *Weigert* first discovered, a necrosis of coagulation has taken place, *i.e.*, the renal epithelial cells, which are otherwise not much changed, have lost their granulation and, above all, their nuclei. This metamorphosis has established itself, because the centre is sustained by adjacent capillaries, although in a limited manner; for, as *Litten* has shown, the renal artery is no end artery in a strictly anatomical sense; on the contrary, collaterals from the capsula adiposa and the ureter exist.

With regard to the *hæmorrhages* which complicate the necrosis behind the arterial obstruction, opinions still differ very much. According to *Cohnheim*, we have diapedesis favoured by disorganisation of the capillary walls (produced by occasional anæmia) and of the smallest veins. This disorganisation of the walls is partly due to a hindrance of the blood current in these vessels, and partly to a retrograde venous current. *Litten*, on the other hand, finds from his experimental observations that hæmorrhages proceed from the collateral arteries; that, similar to engorgement, a mechanical cause exists for the hindrance of circulation, and not a disintegration of the vascular walls. The hyperæmic line of demarcation, found between the anæmic infarctus and the adjoining renal parenchyma, is produced by the irritation of the necrotic tissues on their surroundings.

The final result of renal infarcts is the formation of vascular cicatricial induration of connective tissue by resorption of the necrotic material in consequence of colourless blood corpuscles penetrating and fresh vascular loops organising themselves. Interstitial inflammation establishes itself in the proximity of the defect (which can justify the term metastatic nephritis) and numerous glomeruli and tubuli uriniferi perish and are no more regenerated than in renal wounds (*Mattei*). In such cases where numerous and extensive infarct-cicatrices contract the organ, one also speaks of "embolic contracted kidney".

That occasionally infarct can form itself as sequel to injuries combined with lesion of the renal artery and thrombosis has been shown by *v. Recklinghauser*.

Obstruction of the *main* trunk of the renal artery can cause total necrosis of the kidney (*Cohnheim*, *Friedländer*).

Symptomatology.—The symptoms of renal infarct are very little known, and existence of the affection nearly always causes a surprise at the post-mortem. One may, perhaps, suspect renal infarct when a patient feels *sudden* pain in the renal region accompanied with vomiting and rigor, and especially if the heart is in a weak condition (*Traube*). Generally the urine is normal, but when the infarct is of a hæmorrhagic nature, one usually observes concomitant hæmaturia.

When one suspects this affection, the patient must remain in bed and narcotics be administered.

INFLAMMATION OF THE KIDNEYS (NEPHRITIS).

Classification.—Cliniaters and pathological anatomists have since decenniums sought to single a particular group out of the varied and numerous renal affections. This group is called *Morbus Brightii*, a renal disease remarkable for symptoms of *albuminuria* and *dropsy*. *Bright* was by no means the first to discover this disease, the symptoms of which had been noticed by the physicians of all ages. In 1770 *Cotugno* remarked that albuminuria constituted the intimate and important link between renal disease and dropsy; the observations of the following decenniums leave no room for doubt that certain varieties, such as scarlet fever nephritis and contracted kidney, were known; but this illness is justly named after *the* observer, who was the first to demonstrate by exact investigation the relationship of structural changes of the kidney to dropsy, albuminuria, cardiac hypertrophy, nervous derangements, after *Richard Bright*, who published his memorable treatise in 1827 and 1828. During the following twenty years, confirmatory and, with regard to the symptomatology, more extensive works appeared, especially those by *Christison*, *Osborne*, *Solon*, *Rayer*, *v. Rokitansky*. The latter was the first physician in Germany who made microscopical examination of the kidneys, and who discovered the amyloid degeneration of this organ. The opposition which *Elliotson* and *Graves* (the latter considers albuminuria as the cause of renal disease) brought to bear on *Bright's* theory was without lasting effect.

A second era commenced by the works of *Reinhardt* (1850) and of *Frerichs* (1851), who introduced the theory of diffuse nephritis. On the strength of numerous anatomical-histological researches, they recognised a process which passes through three stages of intensity—*hyperœmia*, *fatty degeneration* and *atrophy*. But further investigations proved that the anatomical lesions of the kidney as origin of Morbus Brightii by no means begin entirely as inflammation; *Traube* differentiated renal disease derived from circulatory derangements and contracted kidney and amyloid kidney from the idea of *Bright's* disease. *Beer* has entered into precise details as to the state of the connective tissue of the normal and pathological human kidney. *Traube* corroborates them and replaces Morbus Brightii by the circumcapsular and intertubular form of diffused interstitial nephritis. The theory of the three stages, already mentioned, confirmed by *Traube*, did not remain undisputed. *Johnson*, on the other hand, on the strength of the study of the alteration of the renal epithelium and of a special interpretation of desquamative nephritis, established together with *Virchow* his theory on the inflammation of the renal parenchyma and made a new classification. This bases itself upon anatomical-pathological considerations and creates in *Bright's* disease very precise distinctions. The first stage of Morbus Brightii responds to the acute, the third to *Johnson's* chronic desquamative nephritis, while its fatty degeneration and non-desquamative renal disease corresponded to the second stage. *Virchow* himself distinguishes a form of nephritis proceeding from the epithelium (catarrhal, croupous, parenchymatous nephritis) and the indurative interstitial form. The most decided opponent of the doctrine of the unity of *Bright's* disease is *Samuel Wilks* (1853), who especially disputes the existence of transitional forms between the large white and the small granular kidney. *Dickson* (1860) distinguishes tubular kidney disease from the intertubular form which leads to granular atrophy. *Grainger Stewart* (1868) recognises inflammation and primary renal cirrhosis as distinct processes. Finally *Rosenstein* regards, in the second edition of his famous manual (1870), parenchymatous and interstitial nephritis, the transitional forms of which *Virchow* has already emphasised, as different types of the same diffused nephritis.

He thus returns to the former mode of classifying *Bright's* disease into different stages, a classification which had already somewhat fallen into oblivion. In the third edition (1886), however, of his book he abandons this theory. Contracted kidney received quite a particular signification through *Gull* and *Sutton's* investigations (1872), which explained the vascular changes already mentioned by *Johnson*, but until then only little known as primary general disease, the contraction of the kidney as partial symptom of the same. This publication received general approbation and a few years afterwards *Bartels'* manual (1873) in its clear and interesting descriptional form attracted in a heightened measure attention and further investigation. The celebrated cliniater decided himself in all important points for the dual doctrine of English exponents, and differentiates the "genuine contracted kidney" as primary interstitial nephritis without previous tumefaction of the organ from parenchymatous nephritis, which can only lead to "secondary contraction". His work was sympathetically accepted in the medical world, but recent investigations however were unfavourable to it. *Weigert* proved (1879) on the basis of precise and methodical pathological and anatomical investigation that the so-called parenchymatous nephritis and contracted kidney exist in no way as sharp and necessarily separate forms, but that the lesion of the parenchyma produces by means of epithelial atrophy an interstitial inflammation. The necessity of the course of Morbus Brightii being in three stages is not proved by *Weigert's* researches, who himself distinguishes four anatomical clinical types of nephritis—an acute, a subchronic, a chronic, and quite chronic form; the latter is represented by granular atrophy. This doctrine, with which we also agree, has found general recognition, as, for example, from *Klebs*, *Cohnheim* and also *Aufrecht*, who, however, does not recognise in the interstitial changes cellular infiltration, but tumefaction of the nuclei of the interstitial tissues, which has latterly been proved by investigations. At the same time one must not forget that after *Rosenstein*, on the strength of the experiments of his pupils *Helferich* and *Bos*, already declared the interstitial and parenchymatous processes as originally simultaneous changes; as *Dunin* had established various deviations from *Weigert's* doctrine, *Fischl* recently negated on the basis of personal observations the epithelial processes as constant initial symptoms of nephritic conditions; according to this author the primary disturbances are generally interstitial and then only follow the affections of the glomeruli and parenchyma, while vascular alterations can follow.

The most ardent advocate of the doctrine of unity is at the present time *Leyden*. Concerning the impossibility of classifying symptoms and establishing clinical forms corresponding with the clearly-defined anatomical disturbances, and recognising the analogy of symptoms of parenchymatous and interstitial nephritis, *Leyden* declares himself a partisan of the unitary theory and only recognises one essential form—*diffused nephritis*, the characteristic of which is an acute inflammation of an acute infectious nature. From a clinical point of view he classifies his diffused nephritis, like *Frerichs*, in three forms. The different forms are only the varieties of evolution of the self-same fundamental type, the idea implied by Morbus Brightii, *i.e.*, that renal diseases characterised by albuminuria and dropsy are not absolutely identical to that of nephritis. In the first place the latter only represents a part of the meaning of the disease, originally recognised by *Bright*, and on the other hand that form of nephritis which never leads to dropsy cannot be termed Morbus Brightii. *Leyden* only excludes from the general view of diffused nephritis the genuine contracted kidney described by *Bartels*, inasmuch as it does not represent the final result of an inflammation, but rather develops itself as a secondary phenomenon, proceeding from a general vascular disease which usually occurs at advanced age; he thus, agreeing with *Gull* and *Sutton's* theory, pleads in favour of a renal *sclerosis*, a term already proposed by *Lecorché*. This renal sclerosis, the different stages and varieties of nephritis and finally amyloid kidney are the only renal affections which really belong to *Bright's* disease.

It must be considered as a favourable sign of a certain conclusion of the numerous questions of the principal features of the pathology of Morbus Brightii that in spite of other, but little important, differences, the same opinion prevailed in the first Medical Congress (1882) between *Leyden* and *Rosenstein*. What *Weigert* did as anatomist, the latter did as cliniater, he was the first who proved the impossibility of *Bartels'* fascinating theory of the difference between parenchymatous and interstitial nephritis. On the other hand *Rosenstein* does not consider dropsy to be a necessary adjunct for the clinical diagnosis of Morbus Brightii. For him the essential fact is in the disturbance of all the constitutive elements of the kidney and especially of the vessels. He distinguishes genuine diffuse nephritis when the organ passes albumin, casts and especially lymph corpuscles. Finally a principal difference between the white and red contracted kidney, in which latter forms vascular alteration plays, as *Ziegler* long ago remarked, an important part, is negated, and *Gull-Sutton's* vascular degeneration is declared to be exceedingly uncommon. The year 1882 brought *Wagner's* consummate manual on Morbus Brightii; it contained a large intrinsic collection of material, but in consequence of its somewhat uninteresting style it did not attract attention in the medical world, and also negates the principle that one or other element of texture is exclusively attacked and establishes the following classification: (1) acute Morbus Brightii, including cholera and puerperal nephritis; (2) chronic

Morbus Brightii and secondary contracted kidney; (3) genuine contracted kidney (granular renal atrophy), and (4) amyloid kidney.

We will omit describing the various classifications which appeared later in interesting works by French and German authors. He who follows their involved history of development soon recognises that it is mostly on a pathologic-histological basis that these difficult researches are made. Together with the desire, however, to make use of the anatomical discoveries with their fine *nuances* as clinical principle of classification, the perception of the impossibility of an agreement of the groups of symptoms noticed at a sick bed, with the results proved by post-mortem, are steadily increased. The cognisance that one is following an, at present, unattainable end, recently rightly condemned by *Giuffrè*, is, as *Ewald* remarks, only *apparently* a *retrogression* but in reality a progress.

We were also, when the first edition of this book appeared (1884), far from the idea of establishing our clinical classification as a histological basis and from distinguishing intertubular and circumcapsular nephritis, etc.; we rather believed that the facts of corresponding classification, which are now so necessary for the physician, might not be separated from the practical grouping in the three well-characterised clinical appearances of disease, *acute* and *chronic diffused nephritis* and *contracted kidney*. Genuine contraction being a non-inflammatory process we exclude it from our observation on nephritis, as also cholera and puerperal nephritis; on the other hand we return to *Christison's* classification in acute chronic forms; we are of *Rosenstein's* opinion that although cases exist where well-characterised stages are to be distinguished, others much more frequently begin as a chronic inflammation without ever having passed through an acute stage. As we place active hyperæmia in the large group of acute diffused nephritis with its deviations and abortive forms, we avoid a separate description. We agree with *Leyden* and *Brault* in considering febrile albuminuria as belonging to the deviations without finding it necessary to place such forms under the classification of Morbus Brightii. Finally, we separate chronic diffused nephritis from genuine contracted kidney (*Wagner*); we urge that this latter group is always characterised by the *degree of atrophy* of the renal parenchyma and that in this group the final stages correspond anatomically with *Leyden's* renal sclerosis (*Bartels*' genuine contracted kidney), which distinguishes itself in its turn by the predominance of vascular degeneration. Thus the group, the type of which is the contracted kidney, embraces the idea, collectively termed by *Ewald* as *atrophied hard kidney*. Finally, in accord with all the modern authors, we placed the amyloid kidney close to these forms.

Thus our classification runs as follows:—

I. *Diffused Nephritis.*
   1. Acute kinds (including active hyperæmia of the kidneys and febrile nephritis).
   2. Chronic forms without decided atrophy.
   3. Chronic forms with decided atrophy and genuine renal sclerosis; completely contracted kidney.
   4. Amyloid kidney.

II. *Circumscript nephritis* (suppurative), *inflammation of the kidneys, nephritis vera.*

Since we first established this classification we have been able to make practical clinical and anatomical observation on a very great number of cases, and we are happy to say that our views become more and more adopted. With regard to details and further investigations we must refer to the special part in recent publications; we must, however, particularly remark that we have met with the "classical course" of the illness much less frequently than we should have expected. We have seen with great pleasure that *Rosenstein*, in the latest edition of his excellent manual (1886), has given up the old classification of nephritis in stages, and has put in its place fundamental groups of acute and chronic diffused nephritis; he has also separately described the contracted kidney as anatomically and clinically well-characterised forms; not, however, in *Leyden's* sense, as the arterio-sclerotic atrophy of the kidney is treated quite apart as an uncommon vascular disease of the organ. Our personal experience also teaches us that primary granular degeneration of vascular origin, which we name *vascular contracted kidney*, and to which *Gull* and *Sutton* attach so much importance, appears to us to be much less frequent than these authors believe. *Rosenstein* confirms our view that the clinical type of the disease cannot be solely based on its mode of development (acute, chronic), but also on the final result of the morbid process, *i.e.*, contraction or non-contraction. On the other hand, we for our part must confess that the etiology, which is generally in pathology of such considerable importance, in this case only plays a secondary part, because every diffused nephritis as a consequence of acute or chronic intoxication of the blood would have to be explained as toxic. We refer especially to micro-organisms, their products of secretion and certain inorganic poisons; because the hæmatogenous origin of Morbus Brightii, according to *Semmola's* theory, who considers the affection of the kidneys as merely secondary, in consequence of certain alterations in the blood serum, has, like all similar theories, so much that is uncertain and unproved, it cannot possibly have a claim on general validity.*

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* In repetition and continuation of his various studies on the dyscrasic (hæmatogenous) nature of *Bright's* disease *Semmola* remarks (*Bull. de l'Acad.*,

We cannot close this part without adding a few words on *Cornil* and *Brault's* new principle of classifying nephritis, based upon their pathological and anatomical researches, a point of view perhaps too little valued by German authors. Based on various observations these authors classify the different forms of nephritis according to their length of duration, but divide the chronic forms into two distinct groups, the one when the changes are irregularly extended over the tissue elements of the kidney (glomeruli, glandular cells, stroma), the other when regularly extended on the tubuli uriniferi and the vessels. In the first case we find the subacute chronic diffused nephritis with the subdivision of glomerulo-nephritis, especially parenchymatous and interstitial nephritis; in the latter the (interstitial) systemic affection of the kidneys or cirrhosis, which can be derived from a glandular (saturnine nephritis) and muscular (granular atrophy) origin. In regarding this theory, which coincides much with English ideas, we must attribute the chief merit to *Bartels*, who contends against the theory of ascribing general value to the different stages of nephritis.

The fact, however, cannot be sufficiently emphasised that our classification also lays a certain constraint on clinical facts in spite of every possible simplification. The groups mentioned have many transitional and mixed forms, and it is by no means uncommon to meet with varieties which connect diffused and circumscript nephritis.

No. 30, 1890) that (1) no parallel exists between seriousness of the diseases of the renal parenchyma and simultaneous albuminuria; (2) that the conversion of the albuminous substances of the blood, together with elimination of non-assimilative albumin, is in itself capable of producing a histologically characterised nephritis; (3) that artificial nephritis can be produced, which, provided the hæmatogenous source of albuminuria be exhausted, does not allow any albumin to pass into the urine. To prove the first point *Semmola* quotes toxic nephritis produced in dogs by subcutaneous cantharadin injections; after the injections had been continued for four weeks the inflammatory alterations of the kidney were very marked, while the albuminuria was slight. In opposition to this condition he places the very severe albuminuria, which takes place in dogs after subcutaneous injections of fowl's albumin. On killing the animal a few days after, the kidneys present no special sign of disease. As standing between these two processes, the symptoms caused by the effect of mercurial salts in animals must be considered. Acute nephritis and severe albuminuria both exist simultaneously. A direct alteration of the constitution of the blood also exists and irritation of the renal elements. *Semmola* proves the second assertion by long-continued subcutaneous injections of albumin. They were applied to a dog for eighty-two days consecutively; at the end of the second month the albumin increased considerably, later serum albumin was also observed, and on post-mortem marked signs of slight nephritis were to be found. The third assertion is proved by a similar experiment. If the injections of albumin were discontinued after forty-five days, albumin soon disappeared, the kidneys showing slight but decided inflammatory changes in the glomeruli and tubuli uriniferi.—W. H. G.

I. DIFFUSED NEPHRITIS.

We must assert the fact that diffused nephritis in the strict sense of the word, that is to say, an inflammation which spreads itself symmetrically over the whole organ, does not exist (*Cohnheim*); in this form of nephritis it is nearly exclusively the blood current which introduces substances into the kidneys producing inflammation of the tissue. As, however, the blood does not flow equally through all renal vessels, nor do all tubuli uriniferi excrete simultaneously, *all* glomeruli cannot be attacked by the inflammation, therefore we must understand by the term diffused nephritis an inflammation of more or less confluent localisations, which are, however, separated from each other by intervals of healthy tissue.

1. Acute Diffused Nephritis.

Acute diffused nephritis answers in the chief points to *Rayer's* acute albuminous inflammation of the kidneys, to *Johnson's* acute desquamative nephritis, to *Traube's* acute hæmorrhagic nephritis, to *Bartels*' nephritis acuta parenchymatosa, to *Lancereaux's* acute epithelial nephritis, to *Lecorché's* néphrite parenchymateuse superficielle ou légère, to *Leyden's* acute nephritis. For the adherents of *Frerichs*' theory it represents the first stage of Morbus Brightii. Finally, the catarrhal or croupous (serious) nephritis described by many German authors belongs to this category.

Etiology and Pathogenesis.—The majority of cases of acute nephritis appear either as secondary manifestations or as complications of other acute illnesses. The most important primary illnesses are :—

(1) *Infectious diseases*, especially acute ones (*Leyden's* acute infectious nephritis). The idea that toxic substances migrated into the body can inflame and degenerate the parenchyma of the organs which principally excrete them is by no means of recent date. But modern bacteriological investigation has taught us to decide on the nature of the toxic substance. Although a great part of the detailed investigation is without intrinsic value, nevertheless the scientific physician cannot dispense with the essential points. Some years ago the medical world accepted certain ideas with great enthusiasm, which attribute the functional derangements and anatomical lesions of the kidneys to the effect of pathogenic micro-organisms. We most decidedly opposed this theory, and demonstrated that, whenever one meets with micro-organisms in the kidney, purulent nephritis is present, and that a specific form of nephritis is never due to the effect of the micro-organisms *themselves*, but is the

result of a toxic substance which is dissolved in the blood passing through the renal epithelium. We were at that time among the first, if not *the* first, exponents of this theory, and showed that, when bacteria was to be found in the organ, *complication* with septic nephritis might exist, and that thus intimate relation exists between acute infectious nephritis and toxic renal inflammation. Recent experiments executed, according to *Gram's* method, prove that kidneys with inflammation of infectious origin can now and then eliminate pathogenic germs, but that these germs by no means can be considered as primary agents of the disease. *Koch's* precise methods have confirmed our opinion, and *Leube*, in proving the absence of bacilli in the non-tuberculous kidneys of consumptives, has given valuable evidence that the human body does not strive to eliminate pathogenic micro-organisms through the kidneys. *Wyssokowitsch* has recently made exact observations as to the fate of micro-organisms injected into the blood, and based on them declares that the kidneys in an intact condition do not excrete bacteria, but that the passage of such bacteria necessitates local lesions in the renal parenchyma. The recent publications of *Neumann's* most conscientious labour agree absolutely with these theories. He rectifies the numerous errors made regarding the ubiquitary bacteria met with in the urine of patients suffering from infectious diseases, and gives evidence to the absence of micro-organisms in the majority of cases of nephritis. It is true that one has observed some pyogenic bacilli to pass the glomerular net (*Schweizer*), and the presence of pathogenic streptococci which distinguish themselves biologically from the common streptococcus in normal urine (*Lustgarten* and *Mannaberg*), but these exceptions, and even the colonies of micro-organisms found by *Babes*, do not suffice to annul the general theory that the origin of *acute infectious diffused nephritis* is due to the action of soluble alkaloids, produced by the elimination of the specific organisms which have occasioned the general malady. In those cases where micro-organisms are to be found in the kidneys we must assume it to be the result of an accidental and secondary invasion.

Of all infectious diseases, *scarlet fever* stands at the head of the list. The number of cases of nephritis which ensue after scarlet fever is greater than any other varieties, therefore this renal disease deserves special description. One must, however, be very careful not to confound scarlet fever nephritis with the albuminuria which appears when the fever is at its height or at the beginning. Scarlet fever nephritis establishes itself with characteristic regularity in the third week as secondary illness (pyrexial albuminuria). This induces the opinion of non-professionals, and even of some physicians, that the little convalescent has become a sacrifice to the disease, because not enough precautions against cold have been taken. The character of the epidemic is generally decisive of the frequency of the disease. In Heidelberg, Thuringia and in Berlin we have observed different types of the epidemic. In some cases nephritis was very uncommon, in others more than half of the patients suffered from it. The much-contended parallelism between nephritis and the severity of scarlet fever, according to the height of fever, exanthema and throat affection, has such frequent exceptions that it is impossible to establish a fixed rule. Slight cases, where even diagnosis is difficult, can produce fatal nephritis, and in severe cases, which even receive no attention or nursing whatever, it does not always appear. This is a fact daily observed. The most intimate relationship may be said to exist between the apparition of nephritis and the intensity of scarlet fever lymphadenitis (*Wagner*, *Leichtenstern*).

Up to the present date hypothesis only exists as to the origin of post scarlatinal inflammation of the kidneys, because of the peculiar character of the disease in establishing itself as a sequel of the primary illness; quite a different character to all other kinds of infectious nephritis.

Scarlet fever nephritis can be said to be a desquamation of the renal epithelium, corresponding to that of the cutaneous epithelium, thus a kind of exanthema. In many cases of scarlet fever the urine contains no renal epithelium (*Bartels*), and the kidney appears (post mortem) quite intact (*Wagner*).

Bohn's mechanical theory is now generally abandoned. According to this theory the inflammation of the kidneys originates as a consequence of renal hyperæmia produced by collateral and compensating fluxion to the organ, and the latter is supposed to be caused by the cutaneous affection which is produced by a double layer of exudata and desquamated epithelium. Nearly all the above-mentioned facts speak against this theory; besides this, in cases of serious cutaneous affections renal inflammation generally appears at the acme of the disease. Thus the theory of a *toxic effect* is the only reliable one. Scarlatinal microbes must be considered as the original source of the soluble poison.

According to *Leichtenstern* the noxious element is seated in the skin at the time of resorption, and thus the toxic substance is introduced into the kidneys. The severity of the renal affections always depends on the quantity of toxic substances resorbed; hence an explanation for the concomitant lymphadenitis and tumefaction of the spleen. We follow this doctrine, but consider the agent which produces nephritis to be a decided modification of the scarlatinal poison. The same experiences in certain cases a transformation and then has a specific action on the renal parenchyma. Of course this transformation does not always necessarily occur. We meet with a remote analogy in the pathology of syphilis. There, also, after the virus has exercised its action in the first and second phase a transformation often occurs which renders it into a new element and produces tertiary varieties. We do not believe that the passage of the virus by way of the kidneys is augmented by the impermeability of the skin in consequence of the scarlatinal dermatitis.

Clinical importance cannot be attached to a type of scarlatinal nephritis closely related to septic renal inflammation; in this form albuminuria, hydrops and uræmia are only of secondary significance (*Friedländer*, *Ashby*). We have observed it in uncommon cases of widely-spread gangrene of the pharynx and suppurative lymphadenitis.

Other acute infectious diseases lead much less frequently than scarlet fever to acute nephritis.

Diphtheria of the pharynx leads to it more often than the others. This kind distinguishes itself from the post scarlatinal form by nearly exclusively appearing in serious cases of diphtheria and corresponding in intensity and duration with the primary illness, and, finally, scarcely ever appearing post diphtheritic. Exceptions, of course, sometimes occur. It is most difficult to precisely distinguish genuine nephritis from simple pyrexial albuminuria, as its intensity is very varied. Sometimes we have most serious forms augmented by stasis and weakness of the heart, caused by croup, sometimes slight forms, so that diagnosis is difficult. Including these latter slight forms *Sanné* believes that nephritic complication occurs in nearly half the number of all cases of diphtheria. *Unruh* has determined that nephritis is the first and only absolutely positive symptom of general infection. The fact has been already mentioned that, based on our own experiments, we refuse to accept the theory of the identity of the contagious agent, producing diphtheria and renal lesion. *Babbe*, *Weigert*, *Wagner*, *Heubner* and *Rosenstein* agree with our view. The theory defended by *Letzerich*, of the destruction of the renal parenchyma by a strong invasion of micro-organisms and their spores, cannot be admitted. The observations of *Oertel*, *Eberth*, *Litten* must be considered as incidental septic complications produced by the immigration of pyogenic microbes.

The etiology of acute nephritis accompanying other infectious diseases is equally obscure. As such infectious diseases we specially mention *abdominal* and *exanthematic typhus*, *typhus recurrens*, *measles*, *small-pox*, *chicken-pox*, *yellow fever*, *whooping cough*, *mumps*, *cerebro-spinal meningitis*, *infections angina* (independent of *scarlet fever* and *diphtheria*), and *pharyngal phlegmon* (*Senator*, *Virchow*, *Landgraf*), *erysipelas*, *croupous pneumonia*, *polyarthritis rheumatica*, serious *infectious enteritis*, especially *cholera infantium* (*Kjellberg*, *Hirschsprung*), *dysentery*, the large group (which includes a number of heterogeneous processes) of *acute infectious febrile jaundice* (*Weil*, *Goldschmidt*, *Wagner*, *Fiedler*, *Nauwerck*, *A. Fränkel* and others), and *other unknown mycosis*, acute infectious *osteomyelitis* (*Mouret*); further, of chronic infectious diseases, *malaria*, *constitutional syphilis*, and *tuberculosis*. In all these illnesses, as in diphtheria, the similarity between simple albuminuria and genuine nephritis occurs. One can, however, generally distinguish the harmless, decidedly more frequent forms of the former, from the clinically more important and uncommon kinds of the latter. A scorbutic state in these acute infectious diseases generally indicates complication with hæmoglobinuria.

Even for this series of infectious nephritis it is certain that renal inflammation is caused by the passage of soluble specific virus, products of the elimination of pathogenic microbes, the existence of which cannot be doubted, although they are in some cases occult to microscopical investigation. These germs often form colonies in the different organs; but in cases where genuine purulent nephritis does not exist they must be considered, similar to the germs of pus and gangrene, as mere secondary agents. *Bouchard*, *Kannenberg*, *Babes* and others have discovered micro-organisms easily to be taken for débris of renal substance and of colouring matter. Their action is not sufficiently proved. It is easily comprehensible that in the course of the infectious maladies just mentioned sources of infectious germs are not wanting.

We have frequently had the opportunity of expressing our opinion on the value of *Letzerich's* investigations and observations on the subject of an acute form of "nephritis bacillosa primaria," which he declares to have observed in twenty-five cases, and which are said to have been produced by stagnating soap water in the gutters. The whole description seems somewhat improbable. One has also described embolisms of micro-organisms analogous to the cocci in pneumonia, met with in primary mycotic infantile nephritis (*Mircoli*). We, as well as *Lustgarten-Mannaberg* have seen similar cases of acute "idiopathic" nephritis, but cannot insist on taking the micro-organisms which we have found as immediate cause of the disease. *Jaksch* raises the same doubts in his recent publications.

(2) Besides acute diseases, the morbid processes combined with *sepsis*, *suppuration* and *inflammation* may be considered as a cause of acute nephritis. It is quite indifferent in which regions of the body such processes develop themselves, be it in the skin, in the bones, in the thoracical or abdominal organs. Amongst the inflammations of septic origin we must specially mention those accompanying *surgical affections*, *puerperal fever* and *ulcerous* (diphtheritic) *endocarditis*. Thirty years ago *Virchow* studied the nature of this "septic nephritis," and *Fischer* some years after made new investigations on the subject. It is sometimes very difficult to differentiate this form from the renal nephritis of the first category, especially with those uncommon, serious acute general infections which have some resemblance to diphtheria (*A. Fränkel*, *Fürbringer*) and to acute exanthema

(*Fürbringer*) and which we hesitate to name simple septic processes and therefore designate as "acute mycosis of unknown origin". It is precisely here that complications with embolic, suppurative nephritis are not at all uncommon. These mixed forms of suppurative and diffused nephritis, so well described by *Litten*, are closely connected with his "bacillary invasions" and *Aufrecht's* "acute parenchymatosis". Without it being possible to establish precise limits, *Aufrecht* himself thought it necessary to add his cases of the above-mentioned kind to *Weil-Goldschmidt's* icteric forms. In short, the correlative relationship of septic renal inflammations is very indefinite, and all classifications are much left to one's own free-will. Of a certain number of renal inflammations one does not yet completely know the nature of the "ichorous substances," (*Virchow*), of the ptomaines (*Brieger*), etc., which in passing through the kidneys produce renal lesions. *Fischer's* opinion that, in cases of furunculosis, butyric acid as product of acid, purulent fermentation, produces nephritis, did not hold good against the recent investigation of the matter by *Robert* and *Rassmann*; in other uncommon cases the renal disturbance appears as a consequence of a colossal invasion of the secretory and circulatory regions of the organ (*Weigert*, *Aufrecht*, *Litten*); finally numerous observations remain, in which the presence is proved of a limited number of micro-organisms in the kidneys. According to our opinion this does not merely suffice for the acceptance of a "renal mycosis," because in a number of apparently quite similar cases the schizomycetes is not to be found in the kidney. We are much more inclined to believe that here also the greater number of cases, where part of the inflammation is not suppurative, is derived from the already often mentioned effect of the poisonous *soluble* secretory products of the micro-organisms. *Virchow* called attention long ago to the toxic character of septic nephritis. It is doubtful whether the streptococcus, which is recently said to play a prominent part as originator of secondary infection in various acute febrile illnesses, influences the development of diffused nephritis. We observed this micro-organism in mycosis of unknown origin and other "septic" renal inflammation, sometimes alone, sometimes only staphylococci and sometimes both kinds of pyogenic germs, and believe that these ubiquitous plebeians of suppuration only settled themselves in the kidney when circumscript inflammatory centres exist with a tendency to suppuration (*cf.* "Suppurative Nephritis"). We attribute special importance to the fact that we have often observed diffused nephritis in cases of septicæmia, where no schizomycetes were to be found in the kidney. *Baginsky* has latterly mentioned similar cases.

We must mention, besides the various surgical affections, under the heading of "aseptic" suppurations, pustulous and ulcerous *skin diseases*, such as eczema, pemphigus, ulcus cutaneum, pediculous impetigo (*Wagner*), scabies (*Scheube*), ecthyma, etc., as cause of renal inflammation. According to *Augagneur*, the course of illness is all the more transient, the less suppurative the skin disease is. Nephritis can also be caused by artificial inflammation of the skin (*Baginsky, Lassar, Salvioli*). In such cases where at the same time medicines are applied externally the case can belong to the toxic type. For renal inflammation produced by skin diseases accompanied by violent pruritus (prurigo, scabies), one must consider irritation of the sensitive nerves as a causal moment (*Scheube*). This interpretation has been suggested by *Wolkenstein's* experiments, who produced albuminuria in animals by faradisation. Finally a good number of cases of diffused nephritis may range with the cases caused by cold. The pathogenesis of acute nephritis produced by *cutaneous combustion* must be considered somewhat complicated. We have already mentioned this in the chapter on "Hæmoglobinuria," but the renal lesion, however, may be said to be caused by inflammation, suppuration and sepsis of the cutaneous surface. The casts, appearing in the urine in consequence of renal irritation produced by wounds and fractures of cylindrical bones, must be considered as of hæmoglobinuric origin. Such cases have also been ascribed to the action of a fibrinous ferment proceeding from coagulations (*Riedel*) or fatty embolisms (*Scriba*).

Finally we must add to these cases those forms of renal inflammation accompanying *heart, pleura* and *lung diseases*. Uppermost on the list is acute and chronic *valvular endocarditis* (*Klebs, v. Bamberger, Wagner, Rosenstein*), the septic forms of which we have already discussed. This form of endocarditis essentially limited to the aorta (*cf.* "Renal Congestion") specially matures genuine diffused nephritis. The intimate connection of this striking fact is still dubious. *Bamberger* calculates nearly twenty-five per cent. nephritic complications in cases of aortic disease, while *Schwalbe* remarked over a third. We agree with *Rosenstein* that acute rheumatic fever in itself is only very seldom the cause of nephritis, but that endocardiac inflammation even of an arthritic organ must be considered as etiological moment. In many cases of phthisis it is hardly to be decided what active part sepsis, suppuration, complicated acute inflammation, hectic fever, tuberculous virus, etc., take in causing nephritis; hæmorrhagic forms were often observed by us here. Perhaps many cases of acute nephritis with tertiary syphilis belong more to this category than to the first-named one, as it is on the other hand clear that cases of "primary" nephritis are often caused by occult suppuration.

Inflammatory diseases of the urinary passages more frequently cause diffused nephritis than one formerly expected. In the majority of cases, however, one generally observes suppurative nephritis or pyelo-nephritis in consequence of the immigration of inflammatory substance by way of the pelvis renalis.

(3) The third group causing the development of acute diffused nephritis is formed by *poisons*. The greater part of the toxic substances (and it is this which increases the practical interest of this group of nephritis) are medicinal substances, especially antiseptics used externally. For this reason it is especially worthy of notice that *Senger* recently on the strength of animal experiments and clinical observations has been able to report thoroughly on the degeneration of the renal parenchyma; he declares that a great number of the deaths occurring after renal extirpation are due to "degenerative uræmia" caused by the use of antiseptics. Individual disposition appears to have the greatest possible influence on the toxic form of renal inflammation. In some cases we observe for weeks the greatest abuse of antiseptics without the slightest injury to the kidneys, in others the smallest quantities will produce sediment. The number of drugs which in some way or another do not injure the kidneys by passing through them is indeed small. The literature on medicinal nephritis so continually increases that we are even forced to put the question, which drug does *not* injure the organ? We cannot enumerate even those toxic substances, the action of which on the renal parenchyma is already proved, which brings forth renal hyperæmia and renal irritation, manifesting itself by albuminuria, and must only limit ourselves to those which produce genuine nephritis. A considerable number even of the latter forms are without clinical importance, because the serious general symptoms and dangerous functional disturbances of other organs rule the situation in the course of the intoxication. In the part treating on hæmoglobinuria we have already mentioned a number of toxic agents some of which produce secondary acute nephritis, thus, especially, *chlorate of potassium.*

Some representatives of the so-called *drastic diuretics* belong to the most important and prevalent renal toxics, especially cantharadin, turpentine, mustard oil, copaiva balsam, scilla and cubebs, possibly also cumol. Of the mineral and organic acids sulphuric and oxalic acid are specially worthy of notice on account of their frequent use. There is also danger for the kidneys in the use of carbolic acid. We have, like *Lücke* and *Rosenstein*, frequently observed intense nephritis after irrigation of the pleura and the intestines, and through dressing with this antiseptic. The much-applied iodoform has similar effects; in fact *Senger* ranges both these disinfectants in his register directly behind the chief poison, sublimate, the effect of which is, according to our experience and that of others, surpassed by no other mercurials used internally. To this drug we must add a series of substances employed in skin diseases. They penetrate through cutaneous excoriations into the blood and lymph current, and thus united in the general circulation produce inflammatory lesion of the kidneys. Although in general their effects remain limited to insignificant disturbances, such as temporary albuminuria and hæmoglobinuria, nevertheless sometimes serious and fatal nephritis occurs. The principal factors of these forms of nephritis of cutaneous origin are frictions of tar, styrax, Peruvian balsam, petroleum, naphthol, chrysarobin, pyrogallic acid, and in fact the divers ointments used against scabies and psoriasis. We have already mentioned that also in some of the cases the alteration of the skin or the resorption of the inflammatory products must be made responsible for the ensuing renal lesion. Much more frequently than commonly admitted salicylic acid and the "mild" boric acid cause inflammatory derangements of the kidney (*Senger*, *Braatz*), while nephritis acuta seldom appears as a sequel to the use of arsenic, phosphor, iodine, lead, when kept within reasonable doses. The last-mentioned drug, however, plays an important part in the etiology of contracted kidney.

Finally, we must add to these agents also chromic acid because of its theoretic interest (like that of cantharadin).

Johnson has recently described some cases of acute nephritis, produced by poisoning from sewer-air, and *Nauwerck* has observed renal inflammation after the use of opium.*

Sharp condiments produce similar effects. *Penzoldt* produced fatal nephritis in dogs by administering ethyl and amyl alcohol.

In conclusion, we must add the inflammatory processes produced in the kidneys by the elimination of toxic substances formed in the organism itself, such as gallic acid in icterus (*Nothnagel*, *Leyden*).

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* *Vachell* and *Paterson*, in a paper read before the Cardiff Medical Society in December, 1893 ("acute nephritis from sewer-air poisoning"), observed that, considering the vast importance of this subject, it is surprising that very little has been brought forward to assist its elucidation. This is no doubt due to our ignorance of the nature of sewer-air poisoning and to the inherent difficulties presented by the evidence at our disposal in its investigation. They describe four cases, three of which occurred in the same house; quantity of urine varied from 20-72 oz. in twenty-four hours, with a specific gravity of 1012-1020, and contained from 1¾-7 grammes of albumin per litre (*Esbach*), blood cells, granular and hyaline casts. Exhaustive inquiry throws some light upon the origin. The absence of scarlet fever is pretty conclusive. Investigation specially directed to this point gave a negative result. Moreover, the patients were examined at a sufficiently early stage in the illness, and also subsequently, to determine the presence of desquamation if it did exist. There was no reason to suppose diphtheria to be at the root of the trouble; in neither patient was there a trace of sore throat at any time, and the early appearance of dropsy in all the cases puts the idea of diphtherial poison out of the question. The mischief did not arise from food poisoning, and no signs of lead intoxication could be found. In short, one is unable to explain the outbreak—which can scarcely be dismissed as a mere coincidence—by causes well recognised, and would suggest that it stands in some relation to the emanations from the sewer ventilator, which was situated in the street directly opposite the house, a few feet from the door, so as to pour its offensive effluvia directly into the house. Definite conclusions can of course not be drawn, as our knowledge of sewer-air poisoning is too incomplete, still *Vachell* and *Paterson's* cases most forcibly draw attention to it and deserve serious consideration.—W. H. G.

In how far certain forms of acute nephritis produced by violent gastritis and intestinal catarrh belong to the toxic form or to the group of infectious nephritis, or finally, to cholera kidney, is difficult to decide. Doubtless various combinations of toxic effect (bacterial ptomaines, divers products of decomposition) and circulatory derangements have to be considered.

As a most fertile cause of acute and diffused nephritis must be reckoned—

(4) *Cold and wet.* Prolonged exposure to dry cold air more frequently provokes chronic than acute nephritis; the latter is generally a sequel to sudden changes of temperature, especially when combined with arduous labour, exhaustion and mental depression, wading through snow, breaking through the ice and other sudden drenchings of the body when heated. If earlier authors (specially *Osborne*) have decidedly exaggerated the frequency of nephritis produced by cold, observations made recently on renal inflammation immediately ensuing exposure to cold (*Dickinson*, *Rosenstein*, *Bartels*, *Henoch*) teach us that we would be too sceptical in considering cold as merely an etiological make-shift. Nevertheless, we must confess that in spite of minute investigations and accurate observations we have never found a case of acute Morbus Brightii, the origin of which could be positively attributed only to cold.

In spite of varied and consecutive study, we must confess to be far from being able to explain how cold is able to produce nephritis. The much-emphasised intimate relationship between skin and kidney is not so easy to explain; recent experiments on renal circulation made by *Cohnheim* and *Roy* sufficiently prove this. They were unable to produce any noticeable change in the circulation of the kidneys by applying either hot or cold water to the cutaneous surface of animals. Hence *Müller's* opinion that increase of blood-pressure, produced in all the internal organs by an augmented contraction of the cutaneous vessels, should be the chief cause of renal inflammation seems rather questionable. The fact that even an enormously increased secretion exercises no particularly great effect on the kidneys speaks against the imputation that antagonism between cutaneous and renal activity is arrested by suppression of perspiration. Finally, earlier and especially English authors (*Todd*, *Johnson*) hold that a pollution of the blood with the secretory products of the skin is responsible for kidney disease. But one does not know if excrementitious substances,. which are thus excreted by the kidneys instead of by the skin, have an inflammatory effect; certainly lactic acid must be excluded (*Frerichs* contra *Fourcault*). Albuminuria produced by artificial suppression of cutaneous perspiration (by covering the skin with impermeable substances, varnish, etc.)

is due to refrigeration through vascular extension and good conduction of heat (*Laschkewitsch*)—recently *Arnheim* stated a decrease of delivery of warmth—and has nothing to do with genuine nephritis. On the other hand, *Lassar* succeeded in producing genuine inflammation of internal organs, and specially of the kidneys, by sudden refrigeration of depilated animals (sudden immersion into iced water).

Perhaps some of the cases of nephritis acuta observed in the course of simple, non-infectious angina by *Laure* and *Lasègue* belong to this category. *Wagner* often noticed the latter affection appear as direct consequence of cold before the commencement of inflammation of the kidneys. *Rosenstein*, however, does not mention cold in his cases.

It must not, however, be forgotten that the causal moment of a considerable number of cases of acute nephritis remains unexplained. We notice again and again such primary forms in perfectly healthy and robust persons, where an apparent etiological reason does not exist.*

According to *Leichtenstern* acute nephritis can appear as an abortive form of various infectious diseases; a secondary inflammation of the kidneys can appear as idiopathic, when accompanying scarlet fever without exanthema and angina.

We will not end this chapter without drawing attention to *Goodhart's* warning, which corresponds with our experience, that we must not regard chronic nephritis exacerbated with hydrops as acute Morbus Brightii. It often happens to our knowledge that the first diagnosis is hæmorrhagic acute nephritis, and that later the case turns out to be genuine chronic nephritis.

Morbid Anatomy.—Great is the astonishment of the student present for the first time at a post-mortem of a patient who succumbed to acute nephritis. In some cases a large reddish-brown swollen kidney appears, in others a pale-buff one; sometimes an organ spotted and lightly speckled, sometimes one with no apparent abnormity. All these facts seem apparently in no connection with the symptoms he has observed during life. In fact a great number of post-mortems are necessary to distinguish fundamental types and varieties, and even now we are always finding pathological processes which do not correspond with the clinical symptoms.

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* *Mannaberg*, *Zeitschr. f. Klin. Med.*, bd. xviii., 223, and *Reck*, *Inaug. Diss.*, Würzburg, 1891, consider a species of streptococcus to be the cause of primary acute diffused nephritis. The former found this kind of bacteria in eight cases out of eleven, the latter in two out of six cases in the urine. Cultures proved that the bacteria found differed most characteristically from other species of streptococci, and minute researches in fifty other cases that they were peculiar to primary acute diffused nephritis, and not to be found in the chronic forms. Injections of the cultures made on animals caused acute nephritis in a space of time varying from one to eight days; the kidneys presented a decided nephritic character, but their parenchyma contained none of the streptococci. *Mannaberg* concludes that the bacteria do not increase in the parenchyma, and that their mere passage suffices to create nephritis. Prognostically this bacterial nephritis seems to be favourable.

Fiesinger's ("Relation d'une épidémie de mal de Bright aigu," *Gaz. de Paris*, pp. 39, 40) account of fourteen inhabitants of a small village of 550 souls being attacked by acute nephritis in the course of eight weeks cannot, according to our opinion, prove the infectious character of the disease, although several cases occurred consecutively in the same house. *Fiesinger* himself remarks that some of the cases suffered from influenza and angina previous to the nephritis, and that these two illnesses were prevalent shortly before.—W. H. G.

Chiefly depending on *Weigert's* description, with which our experience in general agrees, we distinguish, *macroscopically*, two principal types of acute diffused nephritis, both of which are again divided into numerous abortive, mixed and transitional forms.

1. *The large red or variegated kidney.*
2. *The large white kidney.*

The *former* corresponds to the clinical type of *Frerichs'* first stage of Morbus Brightii. The organ is enlarged to sometimes double its size, the capsule is not thickened and readily stripped off, the surface smooth and congested with blood. The cut surface shows venous lumina. The enlargement of the organ is principally due to thickening of the cortex. The same appears nearly always succulent and friable and shows a variegated colouring, inasmuch as greyish-red and pale-buff spots intermingle with deep-red ones. These latter dark-red spots are hæmorrhages which are sometimes so numerous as to give the whole parenchyma a chocolate hue. On careful examination the experienced eye generally discovers malpighian corpuscles, dark greyish-red, and frequently pale granules. The substance is hyperæmic, especially the boundary stratum which often has a dark-red striation. The mucous membrane of the pelvis renalis and calices shows itself catarrhal and slightly hæmorrhagic. Abortive forms of this group are in recent cases very frequent; the kidneys then offer the most varied indication of the already-mentioned characteristics; their normal size is unchanged, and with the exception of the hyperæmic condition. they often appear to the naked eye as healthy organs. It cannot, however, be sufficiently emphasised that such apparently normal kidneys can only be considered abortively nephritic from a distinctly coarse anatomical point of view; for they can present under the microscope disturbances which characterise the most *serious nephritis*, and which must cause *grave functional derangement.*

Our *microscopical* researches have taught us that, as already mentioned, the changes are, with rare exceptions, not really diffused, but generally spread with localised centres over the normal parts of the organ. The hæmorrhages visible to the naked eye are seated especially in the interior of the enlarged cortical tubules and in their loops and collective tubes, more rarely in the malpighian capsules, and in this case the capillary net is compressed. They are less frequently in the intertubular connective tissue. The glandular epithelium appears hypertrophied, obscured by granulation, often crumbled as if corroded on the side towards the lumina; in other parts the degeneration has become adipose. Sometimes we find genuine nuclear atrophy and cellular infiltrations (*Nauwerck-Ziegler's* "hydropische Degeneration"). Capsules and tubules are filled with red blood corpuscles, and here and there with desquamated epithelial cells (desquamative nephritis), round cells, epithelial débris, adhering together and forming shiny and reticular masses; in the loops, collective tubules casts are to be found (*cf.* "Symptomatology").

The parts which deserve particular interest as the locality next attacked by inflammatory agents are the *glomeruli* or malpighian corpuscles; in all decided forms of diffused inflammation they present changes which *appear* to be insignificant but are in *reality* of great importance. *Klebs, Langhans, Litten, Friedlander, Cornil, Waller, Ribbert, Leyden, Wagner, Nauwerck, Fischl, Hansemann* have recently most minutely studied these lesions, infiltration, disintegration, proliferation, desquamation of the convoluted and capsular epithelium, and also peculiar lesions of the glomeruli themselves. Closely connected with this "capsulitis," "glomerulitis," "glomerulo-nephritis," is the abnormal permeability of the glomerular vessels and the transition of albumin and of blood corpuscles into the urine. By applying *Posner's* heat test to the organ we are able to distinguish albumin coagulated between glomerulus and capsule. In certain forms, especially recent ones, the condition is mostly limited to a glomerulo-nephritis, the organ presents to the naked eye a normal condition, so that the diffused acute renal inflammation must be considered as a quantitative augmentation of a glomerulo-nephritis, inasmuch as outside and behind the glomeruli the epithelial cells of the tubules, and, proceeding from them, those of the intertubular connective tissues, are affected. A great quantity of material exists with regard to special structural changes; it is the fruit of assiduous and laborious study with the aid of the most complicated methods. It cannot, however, be our intention to enter here into all the unimportant details, their description being sometimes so exaggerated that distinction between nature and art is often difficult. We rather limit ourselves to a short description of the most important facts, and to our own opinion based on personal observations. Worthy of particular notice are *Friedländer's* observations; he observed under the microscope that the predominant lesions in scarlatinal kidneys were owing to a special alteration of the vascular loops and the glomeruli. The latter are rendered visible to the naked eye by the hyperæmia which is developed in them and resemble greyish white granules; a consequence of the hyperæmia is *increase of volume;* the vessels appear cloudy and opaque in consequence of an inspissation of their walls and the obstruction of their lumina with fine granulated substances together with an increase of nuclei of the cells. *Klebs* was the first who precisely described the alterations of the malpighian corpuscles in cases of lethal scarlet fever nephritis. He already evidenced multiplication of cells in the intertubular connective tissue and also the displacement of the latter. This epithelial proliferation, multiplication of nuclei and desquamation (*Ribbert*) produce deposits round the vascular loops similar to onion skins. The consequences of such conditions are apparent: impermeability of the glomerular vessels by compact obstruction of the lumina or compression of the vascular loops, displacement of the blood current with stoppage of the urinary excretion and its sequels. The changes described by *Friedländer* are said to have the same effect as occlusion of the renal arteries, which can lead to calcification of the epithelial cells analogous to the consequences of ligature of the renal arteries and necrotic condition of the organ observed by *Litten*.

Friedländer's theory, which was originally only adopted for a majority of cases of scarlet fever nephritis, experienced a few years ago quite an unexpected perfection and generalisation through *Langhans*, who, based on his own peculiar technical methods, places the changes of the epithelium in the malpighian corpuscles far behind the lesions of the capillary walls and of the glomeruli, which latter manifest themselves by hypertrophy of the tunica intima and by proliferation. It must be presumed that these vascular processes obstruct the lumina of the vessels with a kind of net, thus causing such serious circulatory troubles as to produce anuria. This net is nearly exclusively composed of granulations formed by the cellular endothelium of the capillaries. *Nauwerck* confirms the existence of these granular masses. For explaining the albuminuria one invokes a different mechanism, abnormal permeability of the glomeruli (that is to say of their walls more than of their cellular epithelium) for certain substances which are generally not easily diffused. The permeability results from the inflammation of the walls. *Langhans*, in fact, does not hesitate to believe that these alterations of the glomerular walls do not alone exist in all cases of secondary nephritis, but also when the cliniater observes predominant albuminous febrile maladies. The lesions of the tubuli uriniferi are in general parallel with the glomerulitis and decide for their part the interstitial lesions; this hypothesis is suggested by *Weigert's* theory, a theory to which *Nauwerck*, however, attributes but little importance. Nevertheless, both *Langhans* and *Nauwerck* most energetically confirm that vascular alteration, nearly exclusively limited to the glomeruli, can alone produce the clinical type of complete acute nephritis and even fatal uræmia. This is epecially the case in scarlet fever and measles nephritis.

Langhans' theory was, however, soon violently disputed, particularly by *Hansemann.* The latter came to the conclusion that the cellular aggregation situated between the capsule and glomerulus originated from cellular epithelium and leucocytosis, but that the multiplication of cellular nuclei in the glomerular loops was far from being a constant condition, and, in fact, was exceedingly uncommon. Thus *Hansemann* considers *Langhans'* descriptions as inexact and only produced by the artifices of preparations, accidental coagulations, etc. *Ribbert* likewise has never observed nuclear multiplication in the capillaries, and believes that *Langhans* has been led astray by the obstruction of the space between the capsule and the glomerulus caused by leucocytes, by nuclei of epithelium situated *outside* the glomerular nets, and especially by the formation of giant cells (a tolerably frequent occurrence) through nuclear proliferation of the epithelium. We ourselves, together with *Friedländer*, have studied the already-mentioned conditions in a great many cases, specially of post-scarlatinal nephritis, and have remarked the more we simplify our technic, the more we make observations on fresh kidneys without submitting them to much preparation, the less we observe capillary obstructions by granular and reticular substance, which he so decidedly argues. *Friedländer*, *Nauwerck* and *Langhans* have frequently shown us their preparations, but we must own that even in febrile kidneys of a moderate degree it has been impossible for us to know if the multiplication of the granular elements has really taken place inside the vascular nets. Besides, it is very difficult to make exact investigations as to their position with regard to the vascular walls because of considerable formation of loops and folds of the capillaries. In all cases it is still impossible to give a decided opinion on *Langhans'* description and to decide whether it is correct or erroneous. Although we refuse to admit that post-scarlatinal nephritis is *always* limited to glomerulitis, we, nevertheless, agree with the modern theories that anatomical lesions of the malpighian bodies are generally the source of nephritis and that they always play an important part in renal pathology. Together with *Ribbert*, *Nauwerck* and *Rosenstein* we draw particular attention to the frequency of the desquamation of cellular epithelium and of defects which result therefrom in the cellular stratum lining the tubuli contorti. On examining a great number of scarlet fever kidneys one does not hesitate to declare that in most cases distinct and extended affections of the glandular cellular epithelium of the cortical stratum, as well as certain alterations of the connective tissue, exist. Now, after *Cornil*, *Brault* and *Fischl's* experiments it is impossible to deny that acute nephritis can commence with other elements than with the glomeruli, and that glomerular affections are not always the first and most marked signs. It is not uncommon that the cellular epithelium of the canaliculi uriniferi, perhaps even that of the connective tissue, can be the seat of the primary lesion, and that they continue to play a principal part during the progress of nephritis. In principle we are of the same opinion as these authors, although the very great part which *Fischl* attributes to the interstitial tissue (as being primarily affected) appears to us somewhat exaggerated, and we also agree with *Langhans* in not accepting *Kelsch* and *Hortolès'* ideas, who considered interstitial affections to be the most important.

Vascular changes do not play a prevalent part in the course of acute nephritis. One has, however, noticed periarteritis in the form of infiltration of the round cells of the tunica adventitia and of genuine neoplastic formations in the connective tissue (*Litten*, *Fischl*). We also allow that it is very difficult to establish a precise limit between the vascular and genuine interstitial process.

Finally, with regard to the *connective stroma*, the interstices of the cortex appear enlarged and infiltrated with round cells, never diffuse, but circumscript, specially where the epithelial cells of the tubuli uriniferi show the most decided changes and round the inflamed glomeruli. In only quite recent cases participation of the interstitial tissues is not to be found, or is hardly perceptible; the parenchymatous affection has not yet affected the connective tissue. Besides the glomeruli and the tubuli uriniferi, *Fischl* and *Langhans* have shown that it is especially the capillaries in the vicinity of the malpighian corpuscles and veins which are affected by the cellular infiltration of the connective tissue. The aggregation of round cells in the interstices can become very considerable in serious cases, voluminous infiltration establishes itself in the vicinity of the capsules and between the tubuli uriniferi with compression of the same, and of the vessels, and even, as we have twice noticed, a kind of nodosity on the surface sometimes occurs (*Wagner's* lymphomatous nephritis).

Large White Kidney.—The second form, the large white kidney, represents a yellowish organ, which is, on section, cloudy, specially on the cortex, on which we find the contours already described somewhat effaced. The chief difference from the large red kidney is the anæmia and the slighter development of hæmorrhages, although they are rarely missing altogether. Under the microscope the cells of the large white kidney are much more adipose and degenerated that those of the large red kidney. This form can be found as well as the hyperæmic one a few days after the commencement of nephritis, and does not therefore necessarily represent a progressed stage. Serious cases with evident adipose degeneration and intense interstitial infiltration are not frequent.

In describing pyrexial albuminuria we shall again meet with the light forms where the kidneys preserve microscopically an absolutely normal aspect. We have already mentioned that various transitions between the two already-mentioned principal forms are very often to be found. We have seen tolerably sanguineous, somewhat hæmorrhagically spotted, otherwise not variegated, kidneys in Thüringia.

We must now consider some peculiar forms of acute nephritis, and such varieties which cannot be well classified in the above-named principal forms; *Leichtenstern's* consistent diffused hyperæmic scarlet fever kidney, with bright, light-red cortex and dark-blue medullary substance, must be mentioned under these forms, likewise a very large colliquative, soft greyish-red kidney, described by *Friedländer* as a "septic hæmorrhagic-interstitial nephritis" met with in scarlet fever as well as in diphtheria, and, according to his theory, not susceptible to the diagnosis intra vitam. *Leichtenstern* found in some cases of scarlatinal uræmia a probably inflammatory acute renal œdema; this either disappears quickly or leads to inflammatory and parenchymatous-degenerated processes of an exceedingly protracted nature. According to *Hortolès* the scarlet fever kidney consists in an acute congestive œdema of the organs. In spite of numerous opportunities we have never been able to meet with conclusive conditions.

According to *Litten*, obstruction and compression of the glomeruli by concentric coils of connective tissue are said to occur already in the third week of scarlet fever nephritis. We are not able to affirm that we have met with this connective link between glomerulo-nephritis and the processes of genuine contracted kidney within such a limited space of time.

Diphtheritic nephritis produces, according to our experience, most frequently a slightly enlarged and anæmic kidney, which to the naked eye appears but little hypertrophied; on examination under the microscope one discovers the above-mentioned changes, lesions of the epithelium of the tubuli uriniferi and of the interstices, as well as initial and decided interstitial changes, slight swellings and desquamation of the capsular epithelium, granular opacity of various glomerular coils; *Cornil* and *Brault* found proliferation of capsular epithelium, *Fischl* fatty disintegration and hyaline degeneration and also the already-mentioned periarteritic changes. *Langhans* has placed diphtheritic nephritis in the group of those renal inflammations in which the lesions are equally limited to the glomeruli and to the canaliculi, and which have a less serious character. According to his opinion, nephritis of moderate and great intensity occurs after pneumonia, typhoid fever, ulcerous endocarditis, puerperal fever and pyæmia.

We have already thoroughly discussed the presence of *micro-organisms* in diseased kidneys, and only mention here that *Langhans* and *Fischl*, like us, have either not found micrococcus embolism in cases of acute infectious nephritis, or, in those cases where they were to be found, they saw the cortex intact or much less affected than the medullary substance. This anatomical condition proves how little schizomycetes have to do with diffused nephritis. We frequently meet with a great quantity of micrococcus embolisms in cases of *septic* nephritis, where they microscopically appear as pale circumscript stripes and spots of a hæmorrhagic nature. For details see chapter on "Suppurative Nephritis". *Litten* describes cases of mycotic renal inflammation with invasion of copious masses of bacteria, which had led to varicose dilatation of the tubuli uriniferi, compression of the capillaries, necrosis and degeneration of the epithelium, so that the distended membrana propria surrounded the bacterial masses and led to the development of micrococcous casts in the papillary stratum.

The forms of *toxic* nephritis which are produced by the poisons causing hæmoglobinuria show various combinations which resemble to a great extent the lesions ensuing after primary or genuine hæmoglobinuria. Other irritative substances represent more the type of *simple active renal hyperæmia* of a frequent pro-stadium of genuine nephritis, as well as *catarrhal nephritis*, the description of which disappears more and more out of the student's manuals. The morbid process characterising this form of nephritis begins at the apex of the pyramids; it principally consists of an epithelial proliferation commencing in the pelvis renalis, and extends itself to the tubuli uriniferi. But, as the source of this nephritis is to be sought for among the catarrhal ones of the urinary passages, we will not enter here into details.

Some forms claim a special anatomical interest. In cases of renal inflammation ensuing through the resorption of *oxalic* acid (experimentally evidenced by *Mürset*) we found hyperæmic organs, the cortical substance of which is striated with white. This condition, visible to the naked eye, is due to the obstruction of the tubuli uriniferi with crystallised oxalate of calcium (*Kobert* and *Küssner*, *A. Fränkel*). We must affirm, after the result of the examinations of a number of kidneys which have been a sacrifice to the poison, latterly so popular for committing suicide, that this striation only appeared in kidneys which were not quite fresh, that is to say, when examined twenty-four hours after death. The crystals have not the octagonal form, but in general that of long hexagonal tablets. Although *Rosenstein* only observes a simple irritative process in these renal affections, we have had occasion to observe that in one case intense genuine nephritis was even accompanied by serious uræmia. That the kidney plays a great part in cases of serious *mercurial* intoxication was first proved by *Cornil*, *Prévost* and *Dahl*, who drew special attention to the deposit of salts of lime in the tubuli uriniferi in cases of interstitial and parenchymatous inflammation. These facts, which *Saikowski*, *Virchow*, *Eisenberg* and others can affirm, have been characterised latterly by *Senger* as the consequences of modern treatment with corrosive mercury, when used as antiseptic in surgical operations; he proved that enormous acute infarction of the organ was capable of filling the tubuli uriniferi with firm masses of lime and of entirely destroying the epithelium. One has met with identical deposits of lime without the least mercurial intoxication (*E. Fränkel*) (*cf.* Nephrolithiasis"), but this fact in no way depreciates the importance of the effects of corrosive mercury in persons poisoned by this mineral. The formation of these lime deposits accompanies the dysentery colics characterising mercurial intoxications. These deposits are rather to be attributed to the decalcification of the bone tissue (*Virchow*) than to the effect of a local necrosis (*Kaufmann*).

In the following part it will be shown how abuse of mercury can produce renal parenchymatous irritation, but limited space does not permit of the detailed description of a series of interesting cases of nephritis experimentally produced, such as cantharadin nephritis (*Stüler*, *Cornil*, *Bos*, *Aufrecht*, *Browicz*, *Eliaschoff*, *Schachowa*, *Lahousse*, *Rosenstein*) with peculiar epithelial desquamation in the tubuli uriniferi and calices, glomerulitis, rapid regenerative processes, interstitial contractive processes, also nephritis produced by intoxication with chromic salts (*Gergens* and *Kabierske*, *Eichhorst*), combined with necrosis of the epithelium (coagulative necrosis), and formation of characteristic casts. For the effects of phosphorus poisoning see chapter on "Degeneration".

According to *Gogli's* examinations of the kidneys of convalescents, the healing process in acute nephritis is produced by genuine regeneration of the glandular epithelium.

Decided anatomical lesions of the pelvis and calices do not belong to our type of nephritis. Diseases found frequently post mortem as secondary alterations in other organs are described in the following chapter.

Symptomatology.—The clinical types of acute diffused nephritis are manifold and much influenced by accessory symptoms, even with regard to the cardinal symptoms: the alterations in the urine and dropsy fluctuate so much that a general view is impossible. We therefore prefer to describe the symptomatology within certain etiological groups in the most simple manner possible.

Scarlet fever nephritis.—The real type of acute infectious nephritis is without doubt *scarlet fever nephritis*.

In the following we will describe its ordinary course in cases of scarlet fever, especially in cases where children are the patients. The little patient is already convalescent and has even returned to its former occupations as "cured" when in the third week of the illness a slight facial œdema usually appears; the child becomes paler, weaker and fatigued, is slightly feverish, vomits and complains sometimes of headache, but more often of pain in the kidneys, which one willingly attributes to distension of the capsule by tumefaction of the organ; finally the child complains of strangury, which is caused by irritation of the mucous membrane of the bladder through abnormally constituted urine, or in consequence of consensual irritability. It is only at this period that one generally remarks that the child passes an astonishingly small quantity of urine, and that small quantity is muddy and varies from a watery flesh colour to decided dark red, and deposits a dirty red and chocolate brown sediment. This sediment consists of blood corpuscles, casts, epithelial cells and undecided débris; on testing one finds a considerable quantity of albumin. Anarsaca increases rapidly and disfigures the already disturbed features and spreads over the trunk, genitals and extremities, giving almost the whole surface of the body an alabaster colouring. Added to this serous dropsy appears; in the course of a few days the little body becomes so swollen as to be unrecognisable; the child lies or sits in an apathetic condition tortured by dyspnœa and coughing, in a state of terror.

Deviations from this course are by no means uncommon. The disease can commence at the end of the first week of scarlet fever, or even in the very first days (*Perret*), but generally it commences in the fourth and fifth week (*Henoch* and *Fürbringer* met with a case commencing at the end of the sixth week); sometimes the disease makes its appearance with serious manifestations; rigor and high fever suddenly colour the urine dark red, it sometimes commences very surreptitiously with normal general health and without fever. *Henoch*, as well as ourselves, has frequently in the course of these apyretic forms met with emphymeral increase of temperature reaching 40°C. (one as high as 41°C.), but without a decided parallel to an increase of the inflammatory process, so that we cannot describe these peculiar abrupt elevations as prognostically unfavourable. We have often seen urine, which was colourless and full of albumin for four or five days, suddenly become hæmorrhagic. Other authors have as well as ourselves noticed polyuria from the commencement as well as non-hæmorrhagic and hæmorrhagic urine alternating. Dropsy sometimes follows albuminuria; it is only seldom absent and then it is in no way a sign of slight cases. *Rosenstein* wisely draws our attention to the fact that the nature of the disease can be mistaken by the physician through the appearance of violent diarrhœa. We have often seen very severe dyspepsia and profuse diarrhœa without the slightest symptom of uræmia.

Urine.—The general condition of the urine in scarlatinal nephritis, the peculiarities of which have been made the subject of special study by *Bartels* and *Thomas*, is, when the illness is at its height, generally as follows: the mean average in the twenty-four hours is considerably *decreased* (only very seldom increased), generally from a quarter to a tenth part of the average quantity; in some very serious cases the patient only passes a few tablespoonfuls during the day, in the most cases complete anuria exists, sometimes for days. The decrease of the blood-pressure produced by inflammation and the rapidity of the current in the capillary loops of the glomeruli, the above-mentioned transplacement of the blood-current inside the latter through the considerable increase of volume of the walls or compression from outside explains the stoppage of the excretion. It appears doubtful whether direct transplacement of the lumina of the glomerular loops by repletion with solid masses plays any part here, all the more as the same vascular walls do not allow of the passage of dissolved blood albumin in considerable quantities. The specific gravity of the urine, which, even when considerably hæmorrhagic, has an acid reaction, is increased, but on account of absolutely decreased excretion of urea seldom to a degree corresponding with the oliguria; it generally deviates between 1020 and 25. By reason of the excessive permeability of the inflamed malpighian corpuscles (according to our opinion specially on account of injury and desquamation of the glomerular epithelium), partly also, as *Lorenz* has recently tried to prove, in consequence of the destruction of the cilia (also studied by *Kruse*), a "protective formation" of the secretory cells of the tubuli contorti, the urine constantly contains *albumin*. The same can occasionally be absent for a short time, even for days (*Henoch*, *Rahnke*), but not during the whole course of the illness, but perhaps a scarlet fever dropsy independent of nephritis exists (as already mentioned) without albuminuria (*Quincke*), which we, however, only consider as cachectic. The quantity of albumin is generally considerable, on an average $\frac{1}{3}$ vol. coagulum, *i.e.*, more than 0·5 per cent., sometimes one and more per cent.; we observed in two cases, one of which was cured, for two days more than $\frac{3}{4}$ vol. nearly two per cent. without considerable admixture of blood, which has by no means a regular effect on the quantity of albumin. The quantity of urea in the urine is nearly always decreased, often to a half and more of the average. The other urinary salts are without clinical importance.

On examination under the microscope we generally find:—

1. *Epithelial, hyaline and metamorphosed granular casts;* these latter are very frequently of a brownish yellow and brown colour, friable in consequence of the imbibition of blood pigment and penetration of débris of blood corpuscles; various transitional forms are nearly always to be met with. We have seen repeatedly in quite recent cases typical waxy, glassy forms; in such cases usually every drop of urine contained a considerable quantity of casts.

2. *Red blood corpuscles* are nearly always found even in pale urine; they appear as numerous discs with irregularly jagged edges, either alone or adhering to casts. They are broken up and sometimes aggregated to blood casts. We have already mentioned that red blood corpuscles originate from the inflamed and abnormally permeable blood-vessels. The fact is, however, worthy of notice that although the urine is hæmorrhagic, the kidney is on section found to be of quite a light pale colour. The blood constituents exist longer in the sediment than albuminuria.

3. *Renal epithelium*, nearly always constant, although not always abundant, either alone or adhering to the casts in so far as it does not itself form the constituent. These cells are frequently enlarged, granular and cloudy. They generally appear as fat globules, sometimes brownish pigmented, and sometimes fused together in a tubular form.

4. *Leucocytes* varying in quantity and sometimes impossible to distinguish from the casts just described, especially when symptoms of fatty degeneration exist. The best way to avoid confusion is to prove the existence of three nuclei by means of acetic acid. We have never seen genuine casts composed of leucocytes.

5. *Detritus*, constant and plentiful, mostly débris of blood corpuscles and epithelium.

6. *Uric acid crystals* and *amorphous urates*, almost always with oliguria, sometimes also with oxalate of lime and hæmatoidin in crystals (*Fritz*) as secondary condition.

7. *Micro-organisms.*

When improvement sets in, the urine becomes gradually free of blood and clearer, increases in quantity, the specific gravity decreasing, in both cases above the average, so that polyuria occurs during convalescence. Blood, casts and albumin disappear slowly, generally only in a few months (*Fischl*); some cases, however, are only completely cured in a year (*Fürbringer*). Slight relapses of hæmaturia occur according to our observation pretty frequently.

Complications.—Dropsy is seldom absent, nevertheless we have often observed its absence in very serious cases. We must mention that it not unfrequently has an inflammable character, especially in cases of scarlatinal nephritis, as *Leichtenstern* remarks. Œdema appears in the form of painful erysipelatous, œdematous tumefaction. This tumefaction sometimes wanders and is serpiginous. Transudation in the serous cavities shows its inflammable nature by pains (peritonitic symptoms, ascites, etc.), and manifests itself post mortem as fibrinous and purulent. Such experiences have, combined with the fact that dropsy precedes nephritis, and can appear in spite of polyuria, and fail in spite of oliguria, caused the acceptance of the theory that dropsy can appear independent of inflammation of the kidneys. Still it seems to us to be somewhat far-fetched considering the generally non-inflammatory nature of the serous transudations to proclaim dropsy as equivalent to nephritis, ensuing from lymph-engorgement in consequence of imbibition of poison. Œdema of the mucous membranes is uncommon, the most dangerous one is that of the aryteno-epiglottidean folds; happily it is an exceptional occurrence. Amongst the various forms of œdema the cerebral and the pulmonary œdema prove most often to be fatal.

Pulmonary œdema has also sometimes an inflammable character (*Leichtenstern's* serous lobular pneumonia).

The disappearance of the dropsical symptoms frequently takes place with copious watery stools; rapid emaciation of the body is remarkable.

Bronchitis, Pneumonia, Peritonitis.—Complications with regular *inflammation* of internal organs, which the body is seldom able to resist, are of great importance and unfortunately exceedingly frequent. How many a child on the high road to recovery is carried away by bronchitis and pneumonia! Acute inflammation of the lungs, sometimes kept in the background by dropsy and uræmia, intra vitam not recognised, often attacks the superior lobules in peracute development. It is not always possible, even post mortem, to distinguish between croupous and catarrhal forms. The frequency of a lobular, non-croupous (desquamative) pneumonia has struck us as well as *Leichtenstern.* It is sometimes impossible to distinguish inflammation of the serous membranes, pleurisy, pericarditis, peritonitis, either clinically or anatomically, from serous dropsy, as will be easily understood from what we have already remarked. Typical peritonitis is rare. We are able to affirm that an operation, specially in cases of acute scarlet fever ascites, can be very dangerous, contrary to in chronic engorged cases (*Oppolzer*). We punctured a boy whose life was in danger from rapidly increasing ascites; the child, however, died from pneumonia in a few days in spite of all antiseptic caution. Post-mortem showed acute peritonitis, which proceeded from the spot of puncture.

Finally, we must mention arthritis, endocarditis (which instals itself insidiously, *Henoch*) and, as already mentioned, severe gastro-enteritis and dysentery with profuse and exceedingly painful diarrhœa.

Valvular and Cardiac Affections. — Until recently lesions of the circulatory apparatus have been somewhat neglected, in spite of the observations of *Traube, Wagner, v. Bamberger,* and *Galabin.* We now know, specially from *Friedländer,* that almost always in the course of scarlet fever nephritis in children, and sometimes quite at the commencement, hypertrophy of the left ventricle, which can increase to double its weight, appears. Also simple acute dilatations in consequence of functional disturbance of the heart muscle are to be found (*Goodhardt*, *Silbermann*, *Steffen*). Decided forms of enlargement of the heart can always be diagnosed during life; slighter forms are often overlooked. According to *Silbermann*, the disappearance of murmur (when present) announces cardiac hypertrophy; we only share this opinion in a limited number of specially febrile cases. The danger of cardiac debility is great in such cases; we have, as well as *Leyden* and others, noticed severe asthma and sudden death as the direct consequence. Above-mentioned author and *Lépine* found a gallop rhythm in fresh cases of the disease, which the latter points out to be a sign of relative cardiac debility, or as a measure of the extent of renal affection. Details on the theory of cardiac hypertrophy are to be found in the chapter on contracted kidney. *Riegel* has latterly drawn attention to the fact that in the majority of cases an increased vascular tension develops itself, always in severe uncomplicated cases, and is combined with a slackening of the pulse. Hypertrophy of the heart appears later, as direct consequence of increased aortic pressure (*cf.* cardiac hypertrophy in cases of contracted kidney). Still we must, in common with *Rosenstein* and *Henoch*, on the strength of a great number of cases, which have been autopsically controlled, most decidedly oppose *Grauer's* opinion, that one always finds hypertrophy of the heart in every case of acute or post-scarlatinal renal inflammation. During the whole course of simple cases increased tension of the pulse is often absent.

Uræmia.—A malicious lurking enemy in this illness is uræmia. Slight forms of this disease are difficult to distinguish from the above-mentioned symptoms; but we are of the opinion that apathy, sickness, obstinate vomiting, headache, pain in the chest, restlessness, dyspnœa, are much more frequently of an uræmic origin than is generally supposed. We agree with *Leichtenstern* that tachypnœa and tachycardia are very frequent and striking symptoms of scarlet fever uræmia. Severe uræmic casualties take the course similar to that already described of eclampsia and coma or in heart-rending exclamations of terror and jactitations and serious asthmatic attacks. Amaurosis, hemiplegia, tetanus and trismus are some of the rarest symptoms. Scarlet fever uræmia can run its course without dropsy, but is almost always accompanied by albuminuria, and without exception by anuria or oliguria. Nevertheless, one has seen serious cases without dropsy, even when the patient has passed no urine for several days. Finally, uræmic accidents can appear as the first symptoms of renal inflammation. We have discussed the theory in another chapter and also mentioned that *Leichtenstern* believes the various uræmic symptoms to be dependent on anæmia and inflammatory œdema of the brain and its membranes (in consequence of the imbibition of scarlet fever poison into the lymphatic passages).

The *course* of scarlet fever nephritis is exceedingly variable. In serious cases the illness usually lasts days and weeks. Unaccountable variations in intensity are frequently noticed. Death results in about one-third of all cases. The transition of scarlet fever nephritis into the chronic form, cases of unusually protracted albuminuria excepted, takes place occasionally, but is very rare.*

We have, like *Henoch*, *Leyden*, *Litten*, *Aufrecht* and others, noticed cases where no doubt could be possible. One must not, however, attribute all cases of contracted kidney which proceed from scarlet fever to the latter. "Why should not persons with contracted kidney have suffered from scarlet fever?" says *Bull*. The special causes of death, which usually take effect at the end of the first week, are generally uræmic coma and serious affections of the respiratory organs, produced by one of the above-mentioned sources. Death often results quite unexpectedly from feebleness of the heart or some unknown reason.

Diphtheritic nephritis.—The form of nephritis during diphtheria is much the same as that following scarlet fever. The former appears in the majority of cases at the acute period of diphtheria, especially in gangrenous forms of this malady. It is seldom that the urine is hæmorrhagic or that dropsy occurs. Anuria and uræmia are exceptional. *Henoch* has never, and we have only once, observed them. Characteristic pathognomonic symptoms are generally wanting, and for this reason the renal affection is so often unrecognised, or disappears behind other symptoms (croup, septicæmia). One often confounds this affection with scarlet fever nephritis, especially when the two maladies appear sporadically at the same time. Even in cases, where scarlet fever exanthema can be decidedly excluded, we could prove the characteristic desquamation at the customary time. The intensity of the excretion of albumin is in cases of diphtheritic inflammation of the kidneys not unfrequently unusually intense; we noticed as much as two per cent. in some cases. Such quantities make the prognosis difficult. A large amount of renal epithelium in fatty albuminous degeneration is to be found in the sediment, red blood corpuscles are not so frequent; casts and cylindroids generally appear in a peculiar condition, quite opaque, but not in consequence of micrococci, but through epithelial débris. We reckon diphtheritic nephritis more distinctly to the desquamative kind. The course of this disease is less variable than that of scarlet fever nephritis. Quick cures of even severe cases are, according to our experience, by no means uncommon. *Leyden* has noticed cases which lasted years and were eventually cured.

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* On the strength of his clinical and pathological anatomical observations on a great number of scarlet fever patients in the Blegdam hospital, Copenhagen, *Sörensen* (*Zeitsch. f. Klin. Med.*, bd. xviii., 298), draws special attention to the fact that the delayed appearance of clinical symptoms is parallel to the acme of the anatomical renal changes. Abortive, slight, severe and very severe cases can be distinguished; but these, however, all constitute *one form*, characterised during the first period by insignificant diuresis, albuminuria and general symptoms, later on by hæmaturia, large quantity of formed elements in the urine, increase of diuresis and disappearance of general symptoms. The illness is nearly always cured and *scarcely ever becomes chronic*. When fatal, the cases are usually complicated with diphtheria or even more frequently with pneumonia.—W. H. G.

Nephritis produced by other infectious diseases.—Acute nephritis in cases of other infectious illnesses is much less known, so that it is difficult to say anything precise as to the frequency of œdema, uræmia and of hæmorrhagic urine, etc. Although certain symptoms vary in different infectious nephritis, they do not deviate much from the clinical type. Their symptomatology is, however, too varied to be united into type of infectious nephritis as *Bouchard* wished.

Amongst the abortive kinds some manifest themselves by renal hyperæmia or catarrh. Some of these kinds have a certain clinical interest.

In cases of abdominal typhus, the renal complications of which have been the most thoroughly studied (the French *néphrite dothiénentérique*), the nephritic symptoms, which generally appear in the second week of the illness, conceal the clinical features of typhus, and sometimes, when the anamnesis is complicated, occasion a wrong diagnosis; hence the renal form of typhus (*Gubler*, *Legrout* and *Hanot*, *Amat*), proofs of which have been brought latterly by *Homburger* and *Wagner*. Our own large experience, made in Berlin, by no means justifies the opinions of these authors; in seven out of eight cases hæmorrhagic nephritis has shown itself from the first to the fourth week accompanied by dropsy, the intensity of which was always proportional to that of the general disease. We have always seen in such cases typhus following its normal course. *Weil* and *Seitz* have also on the strength of absolute precise observations refused to admit of *Gubler's* renal forms. Only once were we surprised by severe uræmic convulsions, which lasted some days, and which really marked the symptoms of typhus, and to some extent justified the term "nephro-typhus". *Longuet* and *Rosenstein* are of the same opinion; the latter declares high fever to be an easy means of distinction. We, however, have lost faith in the favourable prognosis advocated by *Weil*, as in two cases under our observation death was caused by uræmic convulsions, and we agree to a certain extent with *Rénaut*, *Robert*, *Gaucher*, *Capitan*, *Charrin* and *Leyden* in assuming an indefinable predisposition to uræmic complications, an opinion which *Rosenstein* also shares. Complication with catarrh of the urinary organs, as well as with extensive epithelial desquamation, which produces epithelial sediments, giving the urine a purulent aspect (*Wagner*), is worthy of notice.

Measles nephritis, which occurs very seldom (we have only seen it four times) has no marked clinical difference from the post-scarlatinal form. *Henoch* suspects (and certainly he is justified in so doing) that inexperienced physicians often mistake these two kinds.

Rénaut and *Robin* have recently made renal inflammation during the course of small-pox the subject of elaborate study; we have only observed abortive forms in cases of varioloid; the predominant symptom of this nephritis is the presence of a great quantity of blood in the urine, the source of which is more frequently to be found in the pelvis (*Rosenstein*).

Yellow fever nephritis is characterised by fatty degeneration. A very acute course is said to be one of the most important symptoms of this tropical disease (*A. Hausmann*).

A number of recent observations have proved the fact that the usually so harmless *chicken-pox*, which is quite independent of small-pox, can produce genuine hæmorrhagic nephritis, even with dropsy and fatal uræmia (*Henoch*, *Rachel*, *A. Hoffmann*, *Rasch*, *Semtschenka*, *Oppenheim*, *Janssen*, *Wichmann*, *Högyes*, *Unger*).

Scheltema noticed acute renal inflammation with fatal result in cases of *whooping-cough*. We have seen several cases.

Angina, even if combined with scarlet fever and diphtheritic processes, or the already-mentioned severe mycosis, seldom produces genuine nephritis. *Rosenstein* describes some decided cases accompanied by dropsy. We only once saw acute tonsilitis in connection with genuine, and indeed very severe, hæmorrhagic nephritis. We do not agree with *Wagner's* opinion that the secondary phase of syphilis, papulous tonsil-affections, forms a necessary connective link for acute Morbus Brightii: we have seen intense inflammation of the kidneys without noticeable angina.

In cases of nephritis in the course of *febris recurrens* (first described by *Ponfick*) hæmorrhagic urine follows the various attacks with great regularity (*Wagner*). We agree with *Kannenberg* as to the eminently desquamative character of the urinary sediment, especially in this form of infectious nephritis. *Kannenberg* observes numerous schizomycetes in fresh urine; he attributes the origin of the illness to the fact that these germs penetrate into the renal parenchyma; we cannot approve of this theory. According to the latest experiences of *Mommsen*, *Wagner*, *Rosenstein* and *Nauwerck*, acute renal inflammation in the course of croupous pneumonia seems to begin on the fourth up to the eighth day, to remain intense only about a week and in no way to influence the primary disease. *Nauwerck* was able to examine several cases anatomically and in the first place observed an inflammation of the glomeruli of a hæmorrhagic character. According to our own experience we must consider nephritis in cases of croupous pneumonia as a very rare complication, only about two per cent.; simple albuminuria is to be found in one-third of all cases. *Nauwerck* has seen the complication develop itself at the very beginning of pneumonia and even after the crisis. Œdema seems sometimes to be present, although it has not come under our observation. Pneumococci in the urine have little or nothing to do with diffused inflammation. The prognosis is mostly favourable (*Rees*), although transition into the chronic kind has been observed (*Stortz*).

When nephritis complicates *erysipelas*, fever and skin affection disappear quickly; still, one has also observed a sub-acute course with very slow convalescence, indeed even transition into chronic nephritis. We have observed slight forms of nephritis often complicating phlegmonous erysipelas, and consider them as belonging to the septic and pyæmic kinds.

Nephritis following acute *polyarthritis* (*Leyden*) has equally little tendency to become chronic. The same is always complicated with endocarditis, although the presence of the latter cannot always be proved. Serious cases are often accompanied with great loss of albumin (*Bartels*). *Rosenstein* describes two fatal cases with intense œdema. We ourselves have only met with two severe cases of hæmorrhagic Morbus Brightii in several hundreds of cases of acute febrile polyarthritis, and they took a favourable course without dropsy setting in; this shows the rarity of the complication for a town of the size of Berlin. We caused *Schott* to peruse our journals on this subject, and out of more than 1000 cases treated in our hospital the complication has been met with only five times, and with one exception was completely cured. It is possible that even out of this small number one or the other case can be put down to the treatment with salicylic acid. *Riess* has noticed a kind of epidemic of rheumatic nephritis.

Constitutional syphilis seldom leads to regular acute inflammation of the kidneys (*v. Bamberger* only found it four times in about 2430 cases). It takes its course as much with oliguria, high specific gravity of urine, as with the contrary symptoms, in which latter case dropsy is absent (*Wagner*). According to our own observations, slight desquamative forms, with hæmorrhagic indication, appear with secondary syphilis in about eight per cent. of all the cases, especially at the acme of the roseola. We have only twice noticed transition to genuine acute Morbus Brightii in spite of special researches. Slight cases can be cured by mercurial treatment, as *Horteloup* mentions. But the latter method can, as we have already remarked, produce a new toxic form instead of the specific one.

Finally, nephritis in cases of *malaria*, following the most recent observations made by *Kiener* and *Kelsch* in Algiers, is nearly related to scarlet fever kidney; its inclination to becoming chronic and to developing into contracted kidney is very great (*cf.* "Chronic Nephritis"). Albuminuria, intermittent kind, which shows itself during attacks of fever, can be considered as abortive (*Rosenstein, Atkinson*). Paroxysmal hæmoglobinuria as a consequence of malaria is rare. Genuine acute nephritis in a quartana duplicata was described lately by *Rosenstein.* This form seldom ends fatally.

The symptomatology of *septic nephritis* is, by reason of the character of the original illness, difficult to limit. Serious general condition, very high and often variable temperature, quick pulse, dyspnœa, collapse, can just as well proceed from the original illness as from renal disturbances. *Litten* and *Aufrecht* describe interesting cases of mycotic inflammation of the kidneys with serious stomach and intestinal disturbances, rigor, violent pains in the limbs, tumour in the spleen, stoppage of urine, dropsy and, finally, fatal uræmia. Large dichotomic favus casts out of the papillary stratum of the medullary substance have been found in urinary sediments (*cf.* "Pyelo-nephritis"). Death generally takes place in a few days. Nephritis following gangrenous pharyngitis or septicæmia, the cause of which is still unknown, develop themselves in an analogous manner (*Fränkel* and *Fürbringer*).

It is impossible to give a comprehensive statement of the clinical type of acute inflammation of the kidneys in the course of *non-septic suppurations* and *inflammations* (surgical affections, wounds, etc.) in consequence of the want of material. The symptomatology of nephritis proceeding from combustion is difficult to establish on account of the complication of the clinical conditions (see "Hæmoglobinuria"). Renal complications in cases of the already-mentioned inflammatory skin diseases do not appear to offer particular difference from the ordinary type. The independent acute nephritis, which is, according to our experience, distinguished by an obstinate, often periodic, hæmorrhagic course in cases of heart disease (recurring endocarditis), and which is of all the named kinds the most frequently met with at the height of acute rheumatic fever, exercises but small general influence (*Wagner*), as also those forms in cases of chronic pleura and lung disease. In other cases slight general œdema, or quick increase of the already past, or not apparent, slight uræmic symptoms (headache, vomiting), or the nature of urine, found by chance, leads to forming a diagnosis.

Toxic nephritis is often treated as a seldom-observed partial symptom in the symptomatology of intoxication. Nevertheless, fatal uræmia occurs (intoxication with carbonic acid). The impurity of salicylic preparations, administered when the drug first came into use, has, according to our observations, caused many a case of intense hæmorrhagic nephritis with abundant loss of albumin. The drug, which now can claim to be quite pure, can even produce diffuse renal inflammation in sensitive persons. With regard to renal inflammation in cases of sulfoxysmus acutus, which *Munk*, *Leyden* and *Litten* have experimentally and clinically studied, it is important to know that it can appear in a secondary form in the second week, after the first signs of initial renal irritation with lessened urine, very considerable quantity of albumin and products of destruction of red blood corpuscles in the form of casts and amorphous masses (*v. Bamberger*), have already disappeared. Concerning the occurrence of nephritis following treatment for psoriasis and scabies, which *Lassar*, *Unna*, *Kaposi*, *Neisser*, *Wagner*, *E. Lesser* and others have described, it is necessary to mention that *Lassar* observed a fatal case ensuing from inunction with petroleum and was able to show experimentally the alteration in the renal epithelium caused by it (imbibition of fat). Regarding renal lesion produced by *oxalic acid* we must most decidedly oppose the opinion lately expressed by *Rosenstein*, that nothing more than a sign of simple irritation of the kidney is produced. Quite independent of the intense anatomical changes, which we have found in the kidneys of the victims to this intoxication, one woman was cured who had acquired serious amaurosis, complicated with uræmia. The most careful observations of the course of this illness enable us to give the most decided proof of the direct dependence of diffused nephritis on the imbibition of poison. Also in *Fränkel's* case the abundant infarction of the tubuli uriniferi with crystals of oxalate of lime led to a complete stoppage of urine for two days. We have already spoken of the hæmoglobinuric effects of other poisons.

Amongst the renal affections ensuing from intoxication with sharp obsolete diuretics, *cantharadin* nephritis, or, better expressed, cysto-nephritis, usually produces marked clinical symptoms, such as violent strangury, pain in the lumbar region, painful excretion of albuminous coagulum with the urine (fibrinuria). This coagulum can be so voluminous that catheterism is necessary (*Bartels*). Even after several weeks the urine can show blood. *Aufrecht* declares he has noticed a case of this kind develop itself into contracted kidney. Turpentine and copaiva also attack the kidney and the urinary organs.

Acute nephritis produced by catching *cold* generally commences a few hours after the chill is felt with rigor and pain in the kidneys, and develops itself, as we can corroborate, in a few days in a very characteristic manner (*Rosenstein*) into dropsy. In other respects it has great resemblance to scarlet fever kidney. *Bright* and *Dickmann* describe fatal cases. On the whole the prognosis can be considered as an exceedingly favourable one. We have never seen this form develop itself into chronic affections, although we have sometimes observed a fluctuating discharge of hæmorrhagic urine for months.

Diagnosis.—Diagnosis is easy if the disease presents itself in its cardinal symptoms and consists of dropsy or uræmia with excretion of a decreased quantity of urine containing blood, albumin and casts, and when the anamnesis gives proof of the cause of the illness,—difficult, when every characteristic ensemble is wanting. One must try above all to diagnose the disease by exact and repeated tests of the urine. Differentiation from simple febrile albuminuria is generally possible by the proof of a larger quantity of albumin and plentiful morphotic elements in the urine and of its hæmorrhagic condition. We have already discussed the difference of renal hæmaturia from that of the urinary passages. In order to establish the etiological diagnosis of nephritis one has recourse to the quantity of albumin, although the nephritis in question is not connected with an alteration in the blood. When other symptoms are wanting one must examine the nature of the sediment (casts). If the time for observation is short, it is difficult, in spite of exact anamnesis and objective examination, to solve the question, whether the nephritis is primary or only a secondary symptom of another illness. We consider *Rosenstein's* advice as very valuable, not to make every high and long-lasting fever dependent on nephritis, but to seek after particular processes, such as typhus, tuberculosis. The suspicion of a scarlatinal origin can never be exaggerated. One must not forget that nephritis acuta can appear as an abortive form of scarlet fever without exanthema and angina.

Early recognition of infectious nephritis in epidemics, specially scarlet fever, is possible by daily, careful testing of the urine. Those physicians who do not follow this advice will see themselves unpleasantly surprised by the appearance of general dropsy and dangerous uræmia.

In most cases an exact anamnesis will solve the question whether the nephritis is an acute, chronic or recent one, or rather a primary one, or whether it is a fresh outbreak of chronic nephritis; in other cases the question is only solved by examination after the hæmorrhagic symptoms have disappeared; sometimes we have not been able to obtain a decided opinion; we lay, however, in opposition to *Rosenstein*, a great importance on the following signs: hypertrophy of the heart and thread-like pulse; both symptoms are strange to acute and sub-acute nephritis. *Bright's* retinitis is naturally reckoned among the first symptoms to be looked for. Intense hæmorrhagic nephritis, which lasts for months and yields to no treatment, is generally of a chronic origin. On the other hand, fluctuating quantities of blood and casts must not confuse us in the diagnosis of acute, *i.e.*, curable renal inflammation, even when they are noticeable in a moderate quantity for months.

Prognosis.—Apart from the cases in which the general malady is of itself specially dangerous (diphtheria, typhus, pneumonia, urticaria, septicæmia, puerperal fever, poisoning, etc.) the secondary kinds offer an exceedingly favourable prognosis, with exception of the most frequent and most important kind, scarlet fever nephritis. Nearly half of those who have nearly recovered become victims, and it is difficult to understand why some writers reckon scarlet fever nephritis to the favourable kinds. See for course and result of the various kinds the part on "Symptomatology".

The special symptoms so rightly dreaded are: anuria, lasting sometimes for days together, excessive serous dropsy, especially severe uræmia and extensive inflammation of the internal organs. In most fatal cases noticed by us the urine was only slightly or not at all hæmorrhagic. According to *Engelsen* the cases with much blood or slight quantity of albumin give the best prognosis, while in the most severe cases the urine is said to contain but little blood and little albumin. Contrary to the last assertion, we noticed in the greatest number of fatal cases a large quantity of albumin. Numerous lymph corpuscles in the sediment are considered a specially bad sign. One thing is certain, that exactly in cases of scarlatinal nephritis, in spite of apparently unimportant symptoms, death may unexpectedly occur; on the other hand, patients can, in spite of the most serious uræmia, anuria, and pulmonary œdema, still be saved, as we have often had the opportunity of observing. Wide limits must thus be given to the prognosis.

We have already mentioned that, in spite of all possible precautions, acute nephritis may sometimes transform itself into the chronic type. We agree with *Rosenstein* in recommending not to give up hope of eventual complete recovery before the end of a year, and warn against promising a very short course. The duration is generally to be counted by weeks and months, not by days.

Concerning the forms produced by syphilis, we agree with *Wickham* that albuminuria, which appears in the early stage of the secondary period, generally has a favourable prognosis in spite of its pretty frequent connection with œdema, in so far as its disappearance can be traced to a thorough use of anti-syphilitic treatment.

Treatment.—The treatment of acute inflammation of the kidneys will be discussed together with that of chronic diffused nephritis.

APPENDIX.

Albuminuria in Febrile Diseases.

The universally accepted assertions as to the appearance of albumin in the urine in the most varied febrile illnesses date half a century back. The discovery of pyrexial albuminuria is due to *Gerhardt*, who accepted the theory of abnormal processes of filtration under the influence of increased temperature. According to another hypothesis, a relaxation of the vascular walls takes place through altered innervation and a transudation of albumin into the urine in consequence of porosity of the vessels of the kidneys (*Bartels*). *Senator* gives the experimental proof that by a rapid and lasting increase of body temperature albumin can be produced in animals. This writer compares feverish patients without cardiac affections to animals heated artificially. Relying on the experiments of *Zadeck* and *Basch*, *Senator* sees also the cause of albuminuria in the augmentation of the blood-pressure. But we observe "febrile albuminuria" at least equally often at a time in which the paralysing influence of protracted increased temperature and serious general affections of the heart have already reduced the blood-pressure abnormally, and find, on the other hand, by these facts an explanation for pyrexial albuminuria in the depression of the blood-pressure (*Runeberg*), in engorgement (*Körner*), in retardation of the blood-current in the glomeruli (*Leube*).

We are now aware that parallelism between heightened temperature and albuminuria being clinically (*Eckert*) and experimentally (*Mendelson*, *Loewy*) so deficient (the latter can occur with the lowest degree of fever and be missing with the highest one), a direct dependence on the warmth of the body is not to be thought of. Moreover, we are dispensed from the arbitrary acceptation of a sometimes increased, sometimes decreased blood-pressure through the greater probability with which albuminuria in pyrexial illnesses (in general acute infectious diseases and pyæmic processes in the words' widest meaning), according to cases seen by *Becquerel*, *Vernois*, *Virchow*, *Lecorché*, has to be regarded as expression of a *renal* lesion (*Brault: néphrite passagère*), as an "abortive kind of acute nephritis" (*Leyden*, *Eckstein*), which frequently remains thus, and sometimes increases to genuine nephritis. It seems to us that these forms of renal inflammation should be styled a toxic nephritis, as they result from elimination through the kidneys of a toxic agent, *i.e*, the alkaloid excretion of specific micro-organisms. By this explanation the reason is evident why albumin is often missing in febrile affections having no infectious origin (*Stockvis*). Thus we can account for the existence of certain formed elements found in urinary sediment.

The question might arise whether augmented quantity of urea in the blood of fever patients has any influence on albuminuria, but this question is of inadequate importance. *Senator* agrees with *Canstatt* and *Prout* with regard to the alterations by the state of blood in fever.

Anatomical examination usually limits itself to a cloudy swelling (albuminous infiltration) of the epithelial cells of the tubuli uriniferi. The organ is by no means, as we have been able to prove by numerous researches, always hyperæmic. On the contrary, decided anæmia is often evident. We agree on this point with the observations of *Roy* and *Mendelson*, who saw the kidneys of feverish animals become smaller and anæmic through the contraction of their vessels. They explain this contraction by irritation of the central nervous system, produced by infected blood. In advanced cases we find desquamation with formation of casts, hæmorrhages, but only of a slight nature. In other cases the anatomical type of renal catarrh is prominent.

From a clinical point of view no importance can be attached to pyrexial albuminuria. A busy and attentive physician can affirm this daily. It is more rare in children than in grown-up persons (*v. Jaksch*). The urine shows first the character of febrile urine; the quantity of albumin is very slight, and even in the worst cases amounts to only a few milligrammes. *Maurel* found in cases of typhus instead of serum albumin a substance nearly related to peptone, which he considers to be a product of intra-circulatory digestion of blood fibrin; pure peptones were not found by *v. Jaksch* in the course of acute infectious diseases. In the sediment one finds occasional hyaline casts, frequently also blood corpuscles and renal epithelium, occasionally even metamorphosed epithelial casts. Albuminuria generally sets in on the second or fifth day of the illness, and lasts for a number of days. Apparently individual and epidemic factors play a part (*Wagner*).

According to our observations, specially in diphtheria, "febrile albuminuria" thus passes in its anatomical and clinical character so gradually into genuine nephritis that a distinct limit cannot be given. On the other hand, initial scarlet fever albuminuria is so entirely different from genuine scarlet fever nephritis, that "development" of the fever kidney into inflammation is by no means simple. We will not enumerate the various primary diseases; with regard to typhus, we only remark that albuminuria is generally to be found in the course of the second week. Former authors (*Murchison*, *Griesinger*, *Gubler*) noticed it in one-third of the cases. *Frank* decreased this proportion to 15 per cent., and *Rosenstein* to 25 per cent. *Middleton* speaks of 85 per cent., a proportion which according to our observations is decidedly too great. It seems to us that on an average this albuminuria shows itself in 30 per cent. of the cases. In cases of typhus exanthematicus, *Warfvinge* found in 68 per cent. of his numerous cases (1660) albumin. In a similar manner the statements vary as to the frequency of albuminuria in cases of croupous pneumonia (*Crämer, Stortz, Rosenstein*), an illness which, according to our observations (which, however, have not been very systematic), does not so frequently cause decided excretion of albumin as typhus. Concurrent engorgement makes the diagnosis of pyrexial albuminuria much more difficult, specially in cases of croup or diphtheria.

The treatment is the same as that of the primary disease (*cf.* also the treatment of "Acute Diffused Nephritis").

2. CHRONIC DIFFUSED NEPHRITIS

(*without atrophy or marked amyloid degeneration of the parenchyma*).

This form nearly corresponds with the second stage of Morbus Brightii, or with the transition to the third stage, "secondary contracted kidney". Chronic diffused nephritis corresponds to *Wilk's large white kidney*, to *Johnson's non-desquamative nephritis* and to *Lecorché's néphrite parenchymateuse profonde ou grave.* It also represents *Bartels' chronic parenchymatous nephritis*, provided that it be distinct from the amyloid type. It corresponds the most with *Weigert's* second and partially with his third group (sub-chronic and chronic forms of *Bright's* disease), with *Wagner's* chronic Morbus Brightii, except when the form is accompanied by amyloid degeneration or by strong contraction of the kidney. Finally our chronic diffused nephritis is no other than *Cornil's subacute and chronic diffused nephritis* and *Rosenstein's* "smooth or speckled" *contracted kidney.*

As we have already said, recent observations have taught us that one cannot admit *essential* differences between *parenchymatous* and *interstitial* affections. They have proved to us that in chronic nephritis, the kidney can show microscopically the most different aspects, that its colour, volume, consistence can vary considerably, while *histologically* the parenchymatous and interstitial lesions are only different stages of the same kind of affection (*Wagner*). According to this, one observes that with exception of the genuine granular kidney the contracted kidney is only the result of an advanced stage of chronic nephritis. We shall also see that, from a clinical point of view, it is very difficult to distinguish chronic diffused nephritis from the small red kidney, without allowing of a somewhat artificial classification; nevertheless this classification must be kept to, for it corresponds the best with the wants of the practitioner. When amyloid degeneration is accompanied by intense nephritis it appears useless, according to our numerous clinical and histological observations, to classify into separate groups. One must own that if formerly too much importance was attributed to amyloid degeneration, at the present time one goes too far in the other extreme; for the physicians who classify nephritis on the strength of this degeneration have separated entirely similar affections on the strength of a secondary fact, which may only be taken into consideration when it appears with marked intensity.

Etiology and pathogenesis.—In infancy and in old age we meet with chronic diffused nephritis; the third to fifth decennium offers the largest contingent. In some rare cases it originates, as *Bartels* and *Leyden* have proved, from acute diffused nephritis. The etiology of such kinds is therefore analogous to that of the latter; scarlet fever nephritis, inflammation of the kidneys from acute polyarthritis, from erysipelas, from catching cold and (in spite of all opposition) malaria nephritis are specially to be taken into consideration. But the rule that acute nephritis, as such, is either cured, sometimes after months, or ends fatally, and our chronic kind takes its course from the beginning as a *lingering* process, is little changed by the existence of such transitional forms.

It is impossible to give a decided opinion as to its frequency, because a considerable proportion of complication of tuberculosis and syphilis, described by *Johnson* and *Bartels*, is to be considered, according to recent observations (*Weigert*), as amyloid nephritis. Counting the latter kinds, the number of cases which we have noticed within the last three years in the Friedrichshain hospital have doubled themselves, quite apart from the fact that a considerable number are entered under the rubric "heart disease" and "alcoholism". Roughly reckoned one can estimate that out of the number of patients (about 16,000) treated in our wards, two per cent. suffered from chronic Morbus Brightii. This fact, naturally, only expresses the real proportions within certain limits. We wish, however, especially to remark that, contrary to the prevailing opinion, we did not notice a preponderance in men. A special influence of any trade or profession was not to be remarked. On the other hand, climate has a considerable influence.

While German literature has cultivated the opinion that decided anatomical changes of the kidneys are the cause of Morbus Brightii, Italian and French observers have started a theory that *Bright's* disease depends on an alteration of the blood, and that the renal lesion is secondary. After *Graves* and *Elliotson* tried to oppose *Bright's* theory in former times without success, *Gubler*, *Brault* and *Jaccoud*, and especially *Semmola*, have latterly tried to expound an ingenious and hypothetic doctrine, according to which *Bright's* disease represents a general nutritive disturbance, preventing the assimilation of albumin. Urine contains less urea, and consequently the latter remains in the organism; the more the diffusibility of the albuminoid substances increases, these become diffused in the kidneys. The fundamental, perhaps only reason of incomplete assimilation of the albuminoids is to be found in a *progressive disturbance of the respiratory cutaneous functions*, be it that these functions are affected by cold, suppressed perspiration, or various dermatosis. These disturbances themselves are the source of *Bright's* disease, and not elimination of albuminoids by the kidneys with consecutive lesions. *Semmola* draws a parallel between persons with *Bright's* disease and animals whose tegument has been varnished. He invokes in favour of his theory the simultaneous quantities of albumin contained in saliva and bile of nephritic persons, as well as glomerular irritation caused by the continuous passage of albumin in the glomeruli. Similar to *Semmola*, only from a much more general point of view, *Rosenbach* recurs to the idea of his "regulative theory" to the hæmatogenous origin of chronic nephritis or granular atrophy, which is not supposed to represent a primary local kidney disease, but the expression of an absolute increase of the quantity of blood and change of the albuminous corpuscles of the blood; changes in the kidneys, albuminuria, hypertrophy of the heart and polyuria are co-ordinate with this constitutional anomaly. *Sejournet*, *Gaucher* and *Thomas* have developed original ideas as the latest advocates of the chemical origin of our illness. The former urges that Morbus Brightii occurs because the action of the skin has suffered from herpetism (a kind of scrofulous hæmorrhagic diathesis). This herpetism, while suspending the excretion of the perspiration of the skin, would force the different toxic substances which ought to be normally eliminated by it to be filtered by the kidney; hence renal affections. *Gaucher* and *Thomas* invoke another mechanism. According to *Gaucher*, when the oxidation of the tissues is incomplete it forms toxic substances, of which the chief ones are creatin and creatinin, leucin and tyrosin, xanthin and hypoxanthin. When these substances appear in too great a proportion they produce the large white kidney. According to *Thomas* and *Fothergill*, the toxic principle should consist of phosphoric, oxalic and uric acid (*cf.* contracted kidney); they are caused by mental over-exertion, insufficiency of the action of the liver, and by gouty and other diathesis. Nephritic albuminuria of diabetic patients is to be considered here (*Stokvis*).

All these are peculiar theories, some of which appear very plausible, inasmuch as it is possible that toxic substances circulating in the blood and continually flowing towards the glomeruli may introduce lingering inflammation. They are, however, pure hypotheses, equally difficult to refute as to confirm. Certain recent contradictory theories seem nevertheless on certain points against the hæmatogenous theory. *Dessales*, while carefully studying the question, showed how that persons suffering from *Bright's* disease do not eliminate albumin in their saliva, nor in their bile if pilocarpine injections are not made. It is probable that pilocarpine injections have caused *Semmola* to err. *Dockmann* has established for his part the fact that neither blood nor perspiration contains less urea than in the normal condition.

Da Costa, *Longstreth* and *Banti's* theory only deserves to be mentioned from a historical point of view. These authors believe to have found the cause of chronic nephritis in a primary disease of the nervous centres which regulate the afflux of blood to the kidneys (splanchnic nerves, ganglia semilunaria, solaria, renalia, etc.); we will not omit to mention here *Zeroni's* equally arbitrary theory, according to which nephritis albuminurica is a "neuro-vascular" process of illness, which can attract all diseases into its boundaries, the kidneys merely giving the signal. The urea which has passed into the blood produces excretion of albumin by means of an "albumincentrum," which is found in the medulla oblongata.

Concerning the special etiology, authentic causes are wanting for the greatest number of cases. Finally, a number of the causal diseases mentioned in connection with acute nephritis reappear, but in a more chronic form. To this category belong chronic rheumatism, heart disease (*v. Bamberger* calculates for chronic endocarditis seven per cent.), chronic skin diseases (the important etiological part which they play in chronic insidious inflammation of the kidneys has been latterly specially mentioned by *Semmola* and *Duval*) and, finally, suppurative processes of the various characters, be it that the latter affect skin, skeleton or intestines, or that they appear independent as symptoms of constitutional and inveterate syphilis, phthisis and other dyscrasiæ (*cf.* "Etiology of the Amyloid Kidney").

Malaria.—Besides syphilis, *malaria* deserves special mention; the close connection to nephritis has been disputed until recently, but, thanks to the researches of *Kelsch* and *Kiener*, it has been restored to its old proper place; *Rosenstein* corroborates this and declares that typical large white kidney with similar development to acute nephritis is often a sequel to malaria. The neighbourhood in which malaria rages endemically is as important as the character of the epidemic. This is probably the reason why in certain districts this etiology does not exist; as *Frerichs* specially mentions on the north coast (while in Danzig, on the east coast, a fourth of all cases of chronic nephritis can be traced to malaria), and, as *Wagner* mentions, in Leipzig, *Bartels* observed chronic nephritis as a relatively frequent sequel to malaria in Schleswig-Holstein, and *Soldatow* in East Roumelia. *Rosenstein* found it but seldom in Groningen, but remarkably often in South Holland. Finally, *Heidenhain* noticed during an epidemic in Marienwerder hardly a case without secondary nephritis. *Vis-à-vis* of such statements we do not feel ourselves in a position to negate the malaria-genesis of chronic nephritis, and as *Ewald* does, to substitute the influence of low temperature which prevails in those parts. Such a theory must at least prove that taking cold at various parts of the coast must be different. Moreover, *Kelsch* and *Kiener* made their observations in Algiers, which is certainly a favourable climate; they observed Morbus Brightii as frequently there after malaria as in Central Europe; observations made at Bombay show the same result (*Morehead*).

Syphilis.—We owe our thanks to *Mauriac*, *Andronico* and *Jaccoud* for the fullest and newest disclosures on the kinds of Morbus Brightii which arise from *syphilis.*

According to these authors, apart from renal gummata, which have nothing to do with nephritis, each period of syphilis can be united with the different kinds of *Bright's* disease, large white kidney, spotted kidney, granular atrophied kidney.

Cold.—All authors agree that frequently repeated taking cold, especially getting wet, residence in damp, cold apartments, can lead to nephritis; *Rosenstein* even pronounces the frequency of chronic nephritis in the working classes to be due to this fact. Whether these agents are the direct cause of nephritis, or produce rather an increased disposition, we do not know.

Intoxication.—Amongst poisons, *mercury* has, as well as *lead*, been said to be the cause of chronic inflammation of the kidneys, especially when misused in antiluetic treatment; this opinion is apparently false. Our own statistics only show eight nephritic patients out of every 100 under mercurial treatment, and one must even then bear in mind that the renal complaint disappears promptly and completely; doubtless syphilitic renal inflammation is often mistaken for chronic diffused nephritis. The question how far *alcohol* taken in excess tends to induce the disease has been much disputed. On one side *Christison* and *Grainger Stewart* declare that the abuse of alcoholic drinks is the most frequent cause of Morbus Brightii; on the other side, *Dickinson*, *Bartels*, *v. Bamberger*, *Rosenstein* and especially *Cohnheim* believe that it is only a small percentage of chronic alcoholists whose kidneys suffer from the effect. In the first edition of this book, which was published some years ago, we were of *Cohnheim's* opinion, having found post mortem liver-complaint produced by alcohol without the kidneys being affected. But now we are of the opinion, contrary to *Cohnheim*, that alcoholism has undoubtedly great etiological importance. Although we must own that, out of the 500 notorious drunkards who appear each year in our wards in Friedrichshain hospital, the greatest number has no serious nephritic derangements, still there remains a considerable share (more than twenty per cent.) of cases of decided chronic inflammation of the kidneys (mostly tolerably smooth, contracted kidneys), so that we, like *Wagner*, must impute a very considerable etiological importance to alcohol. We especially observe the very frequent co-existence of kidney and liver cirrhosis in the corpse. In private practice it is quite a usual thing to find albuminuria with excretion of casts in innkeepers, brewers and drunkards. These patients also present other symptoms of serious alcoholism and, as it seems, of commencing cirrhosis hepatis.

Finally, a few words on chronic diseases of the *urinary passages.* They are, as *Leyden* and *Rosenstein* rightly remark, by no means rare causes of nephritis. Pyelitis calculosa, purulent cystitis, strictures of the urethra, prostatitis must be especially mentioned. Of course diffused inflammation and granular contraction are more uncommon than the suppurative form or pyelonephritis; nevertheless, we have frequently met with the different kinds combined (even frequently amyloid kidney).

Chronic nephritis is often accompanied by *malignant neoplasms* (tuberculosis and cancer). In such cases we have even seen nephritis so intense that it has totally masked the existence of a neoplasm.

We will discuss the etiological importance of *gout* and *saturnine poisoning* later on.

Pathological anatomy.—The novice frequently quits a post-mortem of chronic nephritis with the greatest disappointment, and, on the other hand, an acute nephritis will post mortem unveil most unexpected conditions. After having witnessed in a great number of cases an almost classical course of the disease, albuminuria, elimination of casts and dropsy, one is surprised at the post-mortem by the greatest variations: large white kidney, sometimes variegated speckled kidney, sometimes an almost normal kidney; in fact one meets with the most startling contradictions with regard to the macroscopic condition of the affected organ. Quite another thing when examined under the microscope. No principle, but more gradual variations appear, which depend especially on the duration of the process, the different degree of hyperæmia and on the degree of degeneration of the lardaceous parenchyma. Seldom only can the existence of the "pathogenic principle" be denied; interstitial processes must be considered as a consequence of epithelial changes.

But to return to the special post-mortem appearances, we generally meet with a tolerably enlarged kidney as the "ordinary" kind of chronic renal inflammation, which usually lasts a year, and has not yet reached the highest stage of contraction. Its capsule is generally readily stripped off, the surface below quite smooth ("smooth contracted kidney"), but not to such an extent as with the acutely inflamed kidney. We sometimes find the capsule adhering firmer, and the surface then appears lacerated and granulated. On section the greyish-red of the corticular substance is found to contain stellate, hæmorrhagic groups and also little yellow whitish (lardaceous) stripes and spots, which give the organ the appearance of being strewed with fine sand (*Bright*). Decided forms appear as the "speckled" or "spotted" kidney (*Rindfleisch*). The speckled form of chronic hæmorrhagic nephritis corresponds with the acute form (*Weigert*). We must consider the latter, however, as a transitional form of the large white kidney. Its occurrence is, according to our experience, very rare. It is comprehensible that if the hæmorrhages are little extended, if the light-coloured spots are wanting, if the surface of the kidney is smooth, and that if, besides, the substance is only slightly changed, these affections can at first sight even escape an experienced anatomist.

In such cases the microscope shows us considerable extension and intensity of the pathological processes. One can see that in chronic nephritis, as well as in acute nephritis, the lesions are not uniformly distributed over the renal parenchyma, but are to be found more or less in circumscript spots. The whole tissue is affected; the malpighian corpuscles, the tubules, the interstitial tissues, even the vascular apparatus usually offers more or less marked changes; indeed it is possible, as *Fischl* and *Schütz* have shown, that glomerulo-nephritis can be complicated with endarteritis and *vice versâ* (*Marchiafava*). We can nearly always prove that the alterations found in the centres correspond to the duration of their development. We find such from the signs of recent inflammation to the atrophic condition. Thus we find the epithelium of the tubuli sometimes larger, cloudier, then much enlarged, sometimes desquamated, sometimes atrophied, even calcified, and in other places considerably diminished and stripped of their epithelial clothing. "Interstitial inflammation" appears in a similar manner, sometimes as cellular infiltration of the distended interstices, sometimes as fibrous connective tissue, wanting in vessels and nuclei. This fibrous connective tissue, together with the tubuli and glomeruli, forms the principal part of the centres of contraction. When these cuniform and radiated centres reach the surface of the kidney, they constitute the retractions and granulations which characterise to the naked eye the first stage of secondary contracted kidney. The alterations of the malpighian apparatus are most varied. In discussing acute nephritis we have already mentioned the affections which characterise glomerulo-nephritis. By reason of the long duration of the morbid process in chronic nephritis (for chronic nephritis usually lasts a year), more or less complete atrophy of the glomeruli prevails. These shrink, their capsules are obliterated, and the glomeruli are, as it were, enclosed in a thick coating. It is, nevertheless, very rare that the whole glomerular apparatus of the kidney is affected. Sometimes the arterial ramifications appear thickened; histological examination proves that this hypertrophy affects the tunica muscularis and interna. Finally, in chronic as well as in acute nephritis, one meets with hæmorrhage, with capsular exudata, and with choking of the tubuli uriniferi with casts. These affections are identical with those described in the acute form, only their multiplicity can engender new phenomena, such as the formation of waxy and fatty casts and fat cells.

Parenchymatous and interstitial inflammatory deposits, recently developed in the cortical substance, produce its tumefaction. On the other hand, it is impossible to discover with the naked eye the degree of atrophy and contraction of the kidney; for hypertrophy of the interstitial elements can to a certain extent compensate for the atrophy of the parenchyma, and it is impossible to perceive the retractive processes. Hyperplasis of the connective tissue can even prevail over atrophy of the parenchyma, and in this case it forms the *large atrophied kidney.*

As yet we have not mentioned a form, which is at the present time frequently discussed; the organ is considerably swelled, soft, has a pale yellow spotted cortex and hyperæmic pyramids, the bluish red of which contrasts very much with the cortex. This is the *large white kidney* of English authors; *Strümpell* proposes calling it large *yellow* kidney, which would be more correct; it is a peculiar kind of amyloid kidney. Next to the light-coloured cortex, its expansion and the size of *Bertini's* columns strike us. Considerable as the microscopic distinction of this organ from the above described "ordinary" chronic inflamed kidney is, still the analogy with regard to histological details in principle is undeniable. *Weigert* showed us that in general the large white kidney only represents a parallel form to chronic hæmorrhagic nephritis, or more correctly a particular kind of this parallel form, distinguished by much greater anæmia and *intense fatty degeneration* of the parenchyma resulting from it. Hence the pale colour, the extended yellow blotches and striation. As to the rest, the structural changes are those of the original form. The glomeruli are not intact, as some authors contend, but always show, although by no means in their entireness, the various changes of the aforenamed kind, which, as we shall see, make themselves remarkable by albuminuria and lessened excretion of water. It must be remarked that, in a number of cases, the changes of the stroma are much less pronounced than the epithelial ones. The latter consist principally of fatty degeneration of the glomeruli, and of the cells of the tubuli contorti. It is not uncommon that the enlarged œdematous interstices only present insignificant cellular infiltration or small fat cells, especially in the vicinity of strongly fatty degenerated canaliculi. This explains the terms so much employed—*chronic parenchymatous nephritis* (*Bartels*), and *inflamed adipose kidney.* Thus one understands how *Kelsch* can deny interstitial affections.

This author considered the form which we studied as the result of necrosis of the epithelium, caused by renal ischæmia. We have frequently met with large interstices of connective tissue impregnated with large fat globules. This kidney resembles the spotted kidney, and seems to be in a state of retraction.

The large white kidney is in its simple non-amyloid form a comparatively rare occurrence. As far as our experience of latter years goes, we must agree on this point with *Wagner* and *Rosenstein*. But if the first-mentioned author distinguishes it from the other kinds of chronic Morbus Brightii, and—what we, based on much experience, do not dare—considers, out of clinical and anatomical reasons, a transition into contracted kidney hardly possible, still one may remark that he himself mentions transitions between the large white and the ordinary variegated kidney. *Rosenstein* distinguishes three varieties of the large white kidney, which we consider to be a useless complication of anatomical classification. Each year's fresh experience teaches us that there are so many transitional and mixed forms, and so many varieties of the already described most frequent fundamental type, specially with regard to cortical swelling, to fat and to hæmorrhagic spots, that it is certainly advisable to reserve the title "large white kidney," for the decided form, where the name "variegated kidney" cannot be applied. The limits between the light-coloured parallel forms and *Weigert's* chronic hæmorrhagic nephritis are more or less arbitrary, if special types do not exist. Insignificant concomitant amyloid does not suffice to place the large white kidney in the group of fatty degenerative kidney, as already described; transitional (or subacute) forms between the large white kidney and acute nephritis are universally recognised.

The retrogressive metamorphosis of the chronic inflammatory processes, which apparently occurs but partially, takes place by resorption of the interstitial exudata in the lymph current and regeneration of the glandular epithelium (*E. Meyer* and *Colberg*). *Golgi* has observed genuine reparatory neo-formation.

The condition of the pelvis renalis is the same as in acute nephritis. *Ackermann* found in one case a lemon-coloured thin mucus in the cavity, which consisted almost entirely of renal casts.

We cannot conclude this part without a few words on the peculiar discoveries which *Formad* made, post mortem, on *alcoholists*. This writer declares that he has almost invariably met with pig-backed, cyanotic, sometimes rough, sometimes atonic, and œdematous organs, the histological, differential character of which, *vis-à-vis* to the engorged kidneys, is specially given in a slighter increase of the interstitial tissues. We have not unfrequently met with such kinds, especially in cases of heart disease produced by alcohol; but it must, however, be mentioned that we have generally found a perfectly normal aspect, and in cases where nephritis existed, much difference from the already described fundamental forms was not visible. The chronic inflamed kidneys of drunkards show very slight, more often anatomical, lesions in the form of spots. When nephritic lesions are observed they are but only slightly different from those of typic nephritis. In alcoholists the kidneys have been for years the seat of chronic inflammation, and generally manifest very small anatomical lesions which appear under the form of spots. Too great importance cannot be attached to the frequency of slight nephritis in alcoholists with circumscribed lesions; when methodically examined under the microscope, the kidneys often manifest the lesions of genuine diffused nephritis. These facts are observed in alcoholists who during their lifetime have never shown symptoms of nephritis, and it is an error that clinical pathological manuals pass over these *abortive* kinds in silence; they do not always degenerate into genuine nephritis, the disorders sometimes remain stationary, and are even partially cured.

We shall treat on other facts found post mortem (dropsy, hypertrophy of the heart, visceral affections) in the following part.

Symptomatology.—It is very seldom that the cliniater finds during the evolution of chronic nephritis, symptoms which enable him to precisely diagnose this or that anatomical lesion. During the last few years we have been able to make post-mortem and microscopical studies of several hundreds of cases, and always found ourselves unable to diagnose on the living the anatomical lesions which we found after death. The character of the urine varies to a great extent on account of dropsy, of the general health and of the course of the malady; the symptoms which accompany the spotted kidney can perfectly well appear in a number of parallel forms, so that a differential diagnosis becomes impossible. The only form which one can somewhat successfully diagnose is the large white kidney; in this case we base the diagnosis on the duration of the malady and the analysis of the urinary sediments. It is naturally to be understood that we except the typical small contracted kidney. For the rest, we can only give the urgent advice to be satisfied with *Bright's* own definition. The cliniater who affirms without restriction the diagnosis of the large white kidney, of the hæmorrhagic spotted kidney, or of complete glomerulo-nephritis, exposes himself to errors; in these cases one can only presume a diagnosis. If we have well understood what *Rosenstein* said in the last edition of his manual, this eminent cliniater agrees with our ideas, and this prudent reserve in the diagnosis, which others consider as decline, is considered by him a progress in the study of nephritis. Nevertheless, we cannot wholly agree with the positive diagnosis of *Rosenstein.*

We recommend great caution before giving a decided diagnosis on any case. What we have said will suffice, we hope, to make the reader understand why we have tried to describe chronic nephritis in grouping the principal symptoms.

Next to the condition of the urine the chief symptom is undoubtedly *dropsy*. We meet with it constantly apart from a decided degeneration of chronic nephritis; it rules the situation and induces the patient to consult the physician, being the first remarkable symptom of illness. We have already discussed its peculiarities. That which distinguishes it from dropsy in cases of acute nephritis is the gradual onset, the slow course. In slight cases, or in such which commence slightly without much clinical importance, it can cause in its generality as anasarca and serous dropsy a considerable degree of danger. Dropsy shows itself all the more, the less chronic the form of the illness, hence its particularly intense development with the large white kidney (we have frequently remarked chylous transudations), as also with those kinds where we miss post mortem decided atrophy of the organ.

A faithful companion of dropsy, *anæmia*, is seldom absent; hæmatometric examinations have shown that the effect is very considerable (*Dickinson*, *Rosenstein*, *Leichtenstern*). The important part played by poverty of blood in the general condition of the dropsical patient is easily comprehensible; serious dyspeptic conditions, tormenting dyspnœa, merciless headache, are not the most unimportant factors in cases of chlorosis and dropsy, and one is only aware of the patient's great weakness when the decrease of dropsy shows thinness of the body.

When decided dropsy and its sequels prove the height of the illness, the urine has generally already for some time given proofs of the serious affection of its excretive organs. It has, in spite of various fluctuations in the daily quantity, become lessened, not to the same degree as with acute nephritis, but still about 1000 ccm., often for weeks at a time not the half of the normal excretion, especially in cases of sub-acute large white kidney, in cases complicated with fevers and dyspnœic conditions it decreases to a quarter and less. Complete anuria is generally absent. A large amount of liquid does not greatly increase the quantity of urine (*Rosenstein*). The specific gravity does not generally vary much (1015-1025), but is relatively decreased. No characteristic reaction exists. The yellow, but seldom decidedly hæmorrhagic urine, when recently passed, shows directly its ominous haziness. The filter separates the two most important substances: *albumin* and *morphotic elements*. At the onset and at the height of the illness the quantity of albumin is always considerable (on an average 0·5 per cent. and more), in fact the greatest quantity of albumin is found in cases of chronic diffused nephritis (*Bartels*). Even before ebullition a thick coagulative magma forms itself, and it is necessary in extreme cases to dilute the urine, in order to prevent the formation of an adherent brown coagulum; these quantities mean four per cent. and more. In a case observed by ourselves (transitional form from the large white and spotted kidney) the quantity of albumin reached, with the enormous specific gravity of 1051 and a daily quantity of about 100 ccm., nearly five per cent.; it rendered, a fact again observed by us, the urine thick and sticky, as if containing sugar.*

Bartels has found by a reliable method the same quantity, *Rosenstein* as the greatest quantity under two per cent. We have *not* been able to corroborate the fact that the large white kidney has on an average larger quantities of albumin than the spotted kidney. Globulin is as frequent as in acute nephritis; the fact of its existence has, according to *F. Hoffmann's* tests, perhaps in so far a practical value as an increase of the albumin quotient (that is to say, serum albumin divided by globulin) is coincidental with the improvement in the patient. Simultaneous hæmialbumose is of no importance.

Microscopical examination of the generally copious sediments shows:—

(1) Always *casts*, generally a large quantity and of all kinds: hyaline, epithelial, metamorphosed in their granular and hard hyaline waxy forms. Shiny oil globules speak as frequently as plentiful casts for the intense lardaceous degeneration of the epithelium, especially in cases of large white kidney (see figure 3), and in those transitional forms of spotted and large white kidney; still we have frequently met with the ordinary spotted or speckled kidney, even when every drop in the bottom of the glass swarmed with fatty casts. The richness of urine in the above-mentioned formations makes its surface fatty and shiny. Free fat in crystalline form often indicates progressed cellular, lardaceous degeneration (*Knoll*).

(2) *Lymph corpuscles*, nearly always constant and in great quantity in cases of nephritis ascendens; they mask nearly all other morphotic elements.

(3) *Red blood corpuscles*. They are inconstant, their quantity frequently varies and only seldom reaches the quantity found in hæmorrhagic urine in acute nephritis.

(4) *Renal epithelium*, generally cloudy or fatty degenerated, even genuine fat cells; what we have said of the fatty casts holds good for the latter.

(5) The unavoidable *detritus*, which moulds itself round the casts, and is difficult to distinguish from fine granular blood pigment and amorphous urates.

(6) *Micro-organisms*. Our experience teaches us that in all

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* In a similar case (specific gravity 1048, quantity *per diem* 280 grm.) I found 4·75 per cent.—W. H. G.

cases where nephritis is not accompanied by an affection of the urinary passage, these microbes denote the onset of urinary decomposition or the presence of vaginal mucus in the urine. For this reason it is advisable to obtain urine from women by catheter.

The quantity of *urea* is much decreased (it is, of course, independent of the excretion of albumin as such), thus the specific gravity of the urine, compared with its quantity, remains below the normal average. *Bartels* and *Tellegen* have shown that the proportion of urea can be normal if the patient is well nourished, that it can in fact considerably exceed the ordinary average. We have also repeatedly found with albuminous diet increased quantity in spite of dropsy. In all cases the proportions of urea have no more diagnostic value than the presence of other urinary salts (*cf.* Symptomatology of Contracted Kidney).

This is the character of urine in cases of chronic nephritis, as far as it exists without serious atrophy of the excretive organs. After what we have said on the subject of acute nephritis, it is not necessary to enter into details as to the dependence of the decreased daily quantity upon the decreased rapidity of the blood-current and the impermeability of numerous glomeruli, or as to the origin of the already-mentioned sedimentary components.

The state of the urine becomes, however, different as soon as the secondary processes of contraction have attained an important position in the chronic inflamed kidney. The influence of a gradual increase of arterial pressure and hypertrophy of the heart then manifests itself. The quantity of urine is more copious, specific gravity and sediment are lessened. Even albumin, although still considerable in quantity, is diminished. It is, therefore, obvious that the forms of chronic nephritis which tend to secondary contraction produce a rather pale, but not very hazy urine, the quantity of which is generally about normal. The more lingering and chronic the process is, in other words, the more the organ succumbs to atrophy and the heart to its sequels, all the more does the urine acquire the pathognomonic character of contracted kidney, which we will treat on in the next chapter.

We cannot conclude without remarking that the chronic inflammatory kidney is very insufficient with regard to the excretion of drugs, such as iodide of potassium, quinine, salicylic acid, etc. (*Chauvet*, *Reale*), and also to the formation of hippuric acid out of benzoic acid (*Stokvis*, *Jaarsveld*, *Kronecker*). A great number of authors contend that the characteristic odours produced by the transition of certain drugs (as, for example, the odour of violets after the use of turpentine) can be absent, and attach a certain prognostic value to the absence; our clinical and experimental observations, as well as those of *Rühl*, contradict this fact.

The inflamed organ itself is scarcely ever the seat of serious trouble. We consider the attempt to prove its enlargement to be useless.

Compared with dropsy and albuminuria, *uræmia* only plays a secondary part in the symptoms of chronic diffused nephritis, while in acute nephritis and genuine contracted kidney this is quite the contrary. According to *Southey's* statistics, in 106 cases of dropsy there are thirty-eight with uræmia. Our own observations hardly show half this number. Possibly the relative rarity of uræmic attacks is due to the retention of urea in the dropsical fluids (*Edlefsen*). This hypothesis is confirmed by *Bartels'* observation. This author, after giving a transpiration bath to a patient suffering from *Bright's* disease, witnessed terrible uræmic convulsions take place as soon as dropsy began to disappear. The complex of symptoms of chronic uræmia is more frequently met with than acute convulsions: stupor, a suffering expression, headache, pruritus cutaneus, frequent vomiting before or after meals or fasting, serious dyspepsia, diarrhœa and specially asthmatic troubles form an ensemble which strongly resembles that which we have known as a consequence of dropsy and anæmia. On the other hand, severe dyspepsia, with or without diarrhœa, can, together with dropsy, master the situation for days and weeks.

A third complex of symptoms, which *Dieulafoy* and *Alibert* consider to be a consequence of vaso-motor affection, consists in hyperæsthesia and of paræsthesia (numb feeling in the fingers, itching, burning, etc.), as one often remarks as the first symptoms of kidney disease. We have only seen these troubles and hæmicrania acquire the importance of genuine neuralgia in cases when the contraction of the kidney is far advanced.

A very important and tolerably frequent complication of the disease is caused by participation of the heart, in form of *dilatation* and *hypertrophy*, especially of the left ventricle. If authors such as *Dickinson* and *Charcot* deny the heart playing a part in this form of chronic nephritis, they are thinking apparently of the parenchymatous large white kidney and amyloid nephritis. The more decided the interstitial processes in the large white kidney are, the more they are complicated with hypertrophy of the heart. According to the statistics of *Galabin* and *Ewald*, who have observed hypertrophy of the heart in about half of the cases of large white kidney, and according to the numerous clinical and anatomical observations by *Weigert*, *Leyden*, *Wagner* and ourselves, it is impossible to deny the important part which such hypertrophy of the heart plays in cases of chronic nephritis of long standing, even before the kidney has reached the real stage of contraction. Although hypertrophy is very marked in cases of contracted kidney, it is but moderate in our form of chronic nephritis, and, when the patient is at the same time dropsical, is only with difficulty to be diagnosed. But occasionally, in the course of time, and corresponding to the increasing atrophy of the kidneys, a decided hypertrophy of the left ventricle of the heart makes itself noticeable in certain cases, even the pulse is the same as in marked cases of contracted kidney. Regarding the mechanism which leads to hypertrophy of the heart, we refer to the next chapter. *Heitler* has recently drawn attention to acute dilatations of the heart which appear suddenly and are of short duration; they are accompanied by cyanosis and dyspnœa.

Retinitis albuminurica, sometimes with hæmorrhages, was already known to *Bright*, and must be considered as a characteristic sign of long duration of the disease. It is to be found as frequently in sub-acute forms as in cases of contracted kidney, and, as *Rosenstein* has shown, without hypertrophy of the heart. Nevertheless, it must be considered as a rule that hypertrophy accompanies retinitis, even to such an extent that certain authors (*Traube* and *Schweigger*) considered it as an intermediary link between renal lesions and retinitis. For details as to the ophthalmoscopic and anatomical condition of the retina, as also the various disturbances of vision which follow, see manuals on eye diseases.

The *ear* is also affected, be it merely nervous, or that real inflammatory appearances form the basis of the complaint. Genuine otitis interna (*Gurowitsch*, *Doumergue*, *Dieulafoy*), pains and noises without palpable material cause (*Tissot*), even sudden deafness caused by affection of the labyrinth (*Downie*, who attributes to this kind the diagnostic importance of *Bright's* retinitis), astonishing changes between paralysis and normal reaction of the nerves of the ears (*Rosenstein*), etc.

The frequency of *secondary inflammatory processes of external and internal organs* in cases of chronic nephritis is considerable, and they are often the cause of death. To this category belong: erysipelas, phlegmon, gangrene, which often proceeds from a more or less œdematous region. One often meets with serious catarrhs, with stomachic and intestinal hæmorrhages (*Henoch*), and with diarrhœa. According to *Hlava* and *Thomayer*, the dyspepsia of persons suffering from nephritis is due to interstitial gastritis, characterised by cellular infiltration of the submucous tissue. These forms of dyspepsia are sometimes quite painless and also accompany other maladies (*Rosenstein*). We have even seen cases where dyspepsia was so intense that its symptoms approached those of cholera nostras or genuine dysentery, and these symptoms mastered the situation to such an extent, that nephritis was only manifested by dropsy and albumin; in some cases it passed unnoticed during life. Together with *Rosenstein* we have remarked that dropsy sometimes remained in spite of violent diarrhœa. We have already mentioned secondary pneumonia (it can even be taken for primary pneumonia) and inflammation of the serous membranes as complications of acute nephritis. According to *O. Meyer*, pleurisy is the most frequent, peritonitis takes the second place and pericarditis the third. When dropsy has already attacked the serous membranes, it is most difficult to distinguish during life, what condition is due to dropsy, and what to inflammatory processes. Beginners are generally surprised with the result of the post-mortem. We know cases in which, even at the post-mortem, it was difficult to determine, what was due to dropsy, and what to inflammation. This is easily to be understood after what has already been said about acute nephritis. The importance of pneumonic processes and serious bronchitis as origins of the most dangerous dyspnœa is apparent.

Liver and *spleen* complaints are frequent (hepatitis, nutmeg liver and lipomatosis hepatis, acute and chronic tumours of the spleen, etc.); do not however usually, provided special signs not existing, exhibit any striking clinical symptoms.

Finally, chronic nephritis causes now and then *habitual abortion* by premature loosening of the placenta (*G. Winter*), or formation of peculiar white nodules in the same (*Fehling*).

The illness can last months and years. It is frequently interrupted by striking remissions and intermissions which are generally deceptive, disappearance of albumin, of urinary deposits, of dropsy and general increase of strength. As a rule, the kinds which begin acutely last about three months, those which commence more gradually have a longer duration. The large white kidney distinguishes itself from the other forms by its onset with dropsy and by the regularity of its evolution, which lasts about a year. Nevertheless, what we have just remarked is far from being constant enough to form the basis of a diagnosis. It is also not a fact that the large white kidney always ends fatally without transition into contracted kidney (*Wagner*). Provided dropsy does not appear, relative recovery or death takes place from one of the afore-mentioned reasons (uræmia, visceral inflammations, extreme weakness, etc.); the "ordinary" kind develops itself generally into contracted kidney.

We cannot conclude the symptomatology of chronic diffused nephritis without a few words on two varieties especially mentioned by *Wagner*. First, on "*chronic hæmorrhagic Morbus Brightii without œdema*," characterised by the fact that during the whole course of the illness, which sometimes lasts over two years, periodic hæmorrhages appear of a few days' duration, and which manifest themselves every week or every month; dropsy is in these cases absent, perhaps because the attacks are so short that it cannot establish itself. In the intervals the urine shows the same character as in cases of contracted kidney. The general condition is little disturbed; important hypertrophy of the heart is missing. We have twice seen cases of this kind which, unfortunately, could only be observed for a few weeks. In one of the cases tertiary syphilis was simultaneous.

The second variety is better known. It is a form of *acute hæmorrhagic nephritis*, appearing during the course of a nephritis which has not manifested very apparent symptoms. This form is about the same as that which *Bartels* describes as an "attack of intercurrent acute nephritis in the course of atrophic renal cirrhosis". *Goodhart* warns against confounding this form with genuine acute nephritis. The anamnesis does not generally afford sufficient proofs for making a diagnosis; in fact sometimes the urine contains no albumin. Usually it is some secondary illness (phthisis), and, according to our experience, often chronic endocarditis, which brings the patient to the physician. During the attacks the urine is either slightly or enormously increased in quantity and is hæmorrhagic. Death, as a rule, occurs pretty soon and is nearly always the consequence of complications; still, we have had such patients in our ward for many months, and we were able to discharge them as improved. Although the urine presents various characters, we do not see in the course of these divers varieties of nephritis the necessity of forming an extra group. We have constantly seen transitional forms which developed themselves into chronic nephritis and complete contracted kidney. Nevertheless, differentiation has a certain practical value. *Aufrecht* only considers *Wagner's* chronic hæmorrhagic nephritis as an inflammatory process concomitant with chronic nephritis; this may be true in some cases, but certainly not in all; we have been able to watch the evolution of the former from the beginning, and we are convinced that it must be considered as a particular form.

We have already mentioned that *abortive kinds* of chronic nephritis exist, and called attention to the practical importance of the knowledge of this fact. One either finds signs of dropsy, anæmia, chronic dyspepsia, etc., or the patient feels and considers himself in good health, and only the microscope reveals to the physician the presence of probably quite circumscribed, but genuine nephritic inflammatory centres (granular casts, renal epithelium).

Prognosis.—When the intensity of the disease is great, death may be considered a rule. It occurs from the already-mentioned casualties or complications before the disease has reached the point of real contracted kidney; this is especially noticed in cases of large white kidney and its similar varieties. Complete cures are the exception; although it is not to be doubted after *Bartels* and *Wagner's* observations, to which we can add two corresponding cases, that even the most serious cases can sometimes be cured. This is especially noticed in such cases in which the causal moments, such as malaria and not too inveterate syphilis, or surgical processes, are accessible to a successful treatment.

But in a considerable number of cases the malady ends with an incomplete cure; one observes long pauses with almost entire disappearance of albumin and dropsy.

In general, intense quantities of albumin, prolonged decrease of the daily quantity of urine, numerous white blood corpuscles in the sediment, are said to be unfavourable signs, while casts, however numerous, do not seem to have much influence on the prognosis. We have, nevertheless, been fortunate enough to notice remissions, even when the patients have had all the symptoms which we have just described. The humane physician should always keep this in mind. When nephritis is already inveterate and one sees that hypertrophy of the heart is developing itself, it proves the evolution into contracted kidney. At the very time in which we are writing there is a patient with a probably very large white kidney who wanders about the hospital gardens without experiencing any pain, and who, a few months ago, had already been twice moribund, and who has remained weeks in uræmic coma, and from whom over fifty quarts of chylo-serous fluid were drawn by puncture. His body, which during the winter was swollen to deformity, now shows distinct signs of renal contraction, and the urine, which generally showed thick masses of coagulated albumin, only now contains a small quantity. We have already mentioned the principal causes of death: collapse of the heart functions, secondary inflammations, general œdema, and finally uræmia. The disease is characterised by the fact that it more often spares weak persons and carries off very vigorous individuals.

Diagnosis.—When renal atrophy is not predominant the essential signs for the diagnosis of chronic diffused nephritis are: great quantity of albumin, the quantity of casts of various forms, white blood corpuscles (naturally these corpuscles lose their importance from a diagnostic point of view when the nephritis is accompanied with catarrh of the urinary organs), fatty degenerated renal epithelial cells, and finally excessive dropsy with characteristic localisation. When all the symptoms are united the diagnosis is easy, taken for granted an amyloid kidney is not present. We have already spoken of the differentiation of engorged kidney. It is not necessary to recapitulate what we have said as to the great care necessary to differentiate all the various symptoms. In order to recognise acute nephritis it will be remembered that the onset is different to that of chronic nephritis. One attaches less importance to the accompanying hæmorrhage, as we have already remarked that chronic nephritis is also frequently accompanied with periodic bleeding. We have seen chronic nephritis with the allures of acute nephritis, with œdema and uræmia, and accompanied till death with hæmorrhagic urine. We cannot warn the physician enough against the tendency of always believing the illness to be acute nephritis whenever he sees violent hæmorrhage (*Goodhart*).

When, on the contrary, the symptoms which we have described as characteristic of acute nephritis are partly missing, or are less frequent, the diagnosis is much more difficult.

The more atrophy there is in chronic nephritis, the more difficult it is to establish the degree of renal sclerosis, especially when the cardiac compensation is deficient. The positive symptoms of complete renal atrophy are as follows: plentiful urine containing small quantity of albumin and sediment and pronounced hypertrophy of the heart.

Treatment of diffused nephritis.—As the treatment of acute nephritis and that of chronic or subacute nephritis seems to be nearly analogous, while the therapeutic indications which concern chronic nephritis with slight atrophy are entirely different from those which respond to the complete contracted kidney, we propose to describe the treatment of chronic nephritis apart.

Prophylaxis.—Nothing is known to us capable of opposing the development of diffused nephritis in the course of acute infectious diseases. We have seen the most dangerous and the most frequent nephritis, scarlatinal nephritis, appear in patients who have been most assiduously nursed, while other patients who have taken no precautions have been spared. It is well known that the general public and also a certain number of physicians attribute the origin of scarlatinal nephritis to catching cold, but *Leichtenstern's* experience teaches us that in cases, when scarlet fever is treated with cold baths, nephritis occurs less frequently; we have never been able to establish with certainty the fact that cold produces scarlatinal nephritis. Nevertheless, we cannot warn too strongly for other reasons (weakness of the heart, etc.) against the systematic use of cold baths, especially in serious kinds of scarlatina where the pulse is quick and thread-like. But if it is impossible to avoid nephritis in infectious maladies, it is not so in toxic nephritis. A physician who knows how to use therapeutic agents with prudence will never have to fear nephritis from their administration.

As to *treatment,* our ever-increasing experience teaches us that it is so far from being as efficacious as certain optimists would believe, and we are certain that the pretended cures of nephritis by certain drugs are only spontaneous. This is especially true in cases of acute nephritis, which, as *Cantani*, *Henoch* and *Rosenstein* remark, has a marked tendency towards recovery. On the contrary we express our astonishment how frequently acute nephritis is cured *in spite* of all the drugs prescribed by certain physicians. We cannot repeat too often that, apart from certain cases where energetic and rapid treatment is necessary, the chief essential consists in sensible hygiene. The patient must, especially if the case be recent, be kept in bed and on a strict diet; in subacute nephritis, as in chronic nephritis, the best auxiliaries of nature are to be found in rational hygiene of the dwelling places and clothes, and in suitable diet.

The *indicatio causalis* can be fulfilled in all forms where malaria, syphilis and especially suppurative processes of all kinds are the cause, and where internal and surgical treatment can be applied. On the other hand, we cannot agree with the now so much accepted treatment of infectious renal inflammation with antiseptics, applied with the intention to kill the pathogenous micro-organisms; we have explained this point at length, and have expressed our doubts as to the part which micro-organisms play in nephritis. One must not forget that, even in cases where renal mycosis is evident, the dose of antiseptics necessary to destroy the microparasites is such a large one, that serious injury to the renal tissues would be produced by it. We are very much opposed to large doses of salicylic and carbolic acid, which can only further injure the already affected kidney. The same is to be said of chlorate of potassium, which has often caused serious renal complications attributed to diphtheria. As to mercury, recommended recently by *Sacharjin* and *Peters*, we counsel it to be only used (and with great caution) in cases where the original illness is syphilis. *Andronico's* theory seems to us somewhat optimistic. Finally, the external use of iodoform is also not without danger. We have discussed the deleterious effect of these drugs in the chapters on "Hæmoglobinuria" and "Etiology of Toxic Nephritis," and do not find recapitulation necessary. The cases in which medicinal poisons have favoured the development of ptomaïnes, instead of arresting them, are more numerous than one believes. Quinine and benzoic acid (*Leyden*, *Klebs*, *Kannenberg*) seem to be preferable to the aforenamed substances, but even these two drugs have never proved themselves to be of great value.

To fulfil the *indicatio morbi* of chronic diffused nephritis is very difficult. Specific treatment against inflammatory renal processes is as yet unknown; but of the number of anti-inflammatory drugs we only have the *astringents*, already used by *Bright* and *Frerichs*. One knows that these authorities administered them with the intention of producing the normal tonus of the renal vessels. *Ribbert's* recent experiences have confirmed *Bright's* theory. But our observations have only given us negative results. Tartar emetic and nitric acid have been recommended as antiphlogistic remedies, but our experience teaches us that they are not advisable. It is certain that they cannot be used without causing some harm to the renal tissues. Derivatives applied to the lumbar region are useless, and one must forbid vesicatories; finally, the application of ice has very problematical effects. Cupping applied in the lumbar region has the advantage of relieving the pain for a moment. It is preferable to make use of dry cupping; bleeding increases the anæmia.

It has been suggested to modify the circulation and to prevent the fluxion of blood towards the kidneys by heating the skin, and thus producing hyperæmia, but the results are as yet not sufficiently known. Let it be as it may, we must not forget that all organs under recent inflammation exact complete repose, and that, when a patient is attacked by nephritis, the physician must at once prescribe absolute rest. When the nephritis has passed into the chronic state the question is quite different. Is one obliged to keep the patient in bed? Some physicians do not hesitate to reply to that question in the affirmative. It is evident, that if the patient suffers pain, if he is exceedingly feeble, or if he is menaced with inflammatory complications, repose becomes imperative; but if the strength is tolerable, dropsy only slight, and one foresees atrophy of the organ, we do not consider it necessary to deprive the unfortunate patient of short walks in the fresh air when the weather is favourable, even at the risk of increasing the albumin by movement. We were witness of a case which is doubtless an exception, but not the less instructive. A patient with *Bright's* disease, who had been kept in bed for months, suffered so much from the continual inactivity that he ran away from the hospital in the middle of summer. He went into the forest and perspired to such an extent that his dropsy disappeared; this patient, whom we believed to be dead, returned to us a few months later cured.

According to *Cantani*, friction with oil has a favourable effect on the curative hyperæmia of the skin.

When the temperature of the patient increases, as one notices sometimes in acute nephritis, one must in general avoid antipyretics. If the patient complains of a burning heat, one can try a little quinine, or antipyrin or antifebrin, but never digitalis or salicylic acid. These drugs can have at best a purely subjective effect, objectively they do more harm than good. Lukewarm baths can be tried; these baths never harm, and have a soothing effect (*Wagner*). Slight cases have a decided tendency to spontaneous recovery; it is preferable to treat them expectatively. The patient should be kept in bed, and be given light diuretics (weak tisanes, effervescents, etc.); in case of constipation, mild aperients. The strength must be kept up with milk, nourishing vegetable soups (leguminosis) and light nourishment. Nitrogenous diet is not necessarily forbidden.

In serious cases rational fulfilment of the *indicatio symptomatica* sometimes produces good effect. Such indications are as follows:—

(1) *Decrease of urinary water.*—When a person suffering from *Bright's* disease is not too rapidly attacked by œdema and his digestive functions are not troubled, it is advisable to give him plenty of water, milk, lemonade, and effervescents, and in such cases it is seldom that the albumin increases. *Glax's* theory, that

decrease of liquid ingesta increases the diuresis, has always been incomprehensible to us. Real diuretics should be applied to cases accompanied by dropsy and uræmia. We have seen inexperienced physicians confound anuria with retention of urine, and even attempt to use the catheter. Care must be taken to avoid such errors; we once saw serious cystitis develop itself in such a case.

(2) *Albuminuria.*—Especially in cases of chronic nephritis, and when accompanied with intense loss of albumin, this symptom should be the object of special treatment. We have often seen physicians, and even good cliniaters, careless of the *régime* which they ordered for their patients; they have even prescribed, after nephritic albumin has been proved, a diet containing as much albuminous matter as possible, such as meat, eggs, and that without taking the trouble of ascertaining the quantity of albumin excreted. This is wrong. On the other hand, we cannot advocate *Senator's* principle in forbidding eggs altogether, and only prescribing food without albuminous matter. We have shown that eggs occasionally increase the proportion of albumin, but the loss of albumin is amply compensated for by these viands themselves; and as it has by no means been clinically proved that the excretion of albumin is proportional to the degree of inflammation and that nourishment containing albuminous matter irritates the kidneys, we do not see why patients should be deprived of such precious forms of nourishment. When albumin keeps increasing for a great length of time, when a patient, for example, loses ten grammes of dry albumin daily for weeks, it is evident that his nutrition is singularly compromised, and that his life itself is in danger. In cases of this kind we do not hesitate to give the patients nourishment containing as much albumin as possible, and we prescribe as much as his digestive organs can support; we have always found this nourishment agree with our patients, and the quantity of albumin has rarely increased. It is of much greater importance to keep up the strength and general health of the patient with nitrogenous food than to try to avoid a slight increase of albumin (*Rosenstein*). It has even been noticed, that, when the body does not receive sufficient albuminous matter, the albumin increases (*Sehrwald*). There is only one case where we recommend meat to be entirely forbidden, and that is in cases of recent nephritis in full development, such as frequently met with in infantine scarlet fever nephritis. In fact, according to the experiences of *Senator*, *Henoch*, *v. Dusch*, ourselves and other experienced cliniaters, nitrogenous nourishment in such cases does not only produce an increase of albumin, but gives at the same time such a spurt to the inflammatory processes that the hæmorrhagic deposits and the casts increase and the general state of the patient becomes worse. *Penzoldt* has given practical proofs of

this. In cases of chronic nephritis we successfully prescribe meat and meat essences. *Rosenstein* and *Cantani* do the same in spite of *Semmola* and *Gaucher*, whose opinions on the dependence of nephritis on beeftea, essences of meats, meat powder and other "poisons" we have already mentioned. Naturally, in such cases where the patient suffers from intense dyspepsia, which, together with the loss of albuminous matter, is the principal cause of hydræmia and anæmia, one must be very careful; it is advisable to follow *Senator's* advice and let the patient eat little but often. *Potain* and *Semmola* recommend absolute milk-diet, *Schmidtlein* considers this *régime* anti-dropsical and *Jaccoud* as a preventative; but we have, unfortunately, seen more than once that dyspepsia prevented patients keeping to a milk-diet, even when brandy or coffee was added. The more our practice increases the more we see patients incapable of standing milk-diet, and when the cessation of this *régime* is a veritable relief. *Oestreich* declares that he has seen *one case* where milk-diet entirely banished albumin, but this one observation does not suffice to make us change our opinion on the subject. *Sobotta* has shown that the lactic *régime* does not produce a sensible diminution of albumin, although this diet exercises an excellent influence on nutrition. Nevertheless, one must not omit milk in the patient's nourishment;* on the contrary, it should be always given with the other food. But one of the principal forms of nourishment is leguminous substances; these excellent preparations of *Liebe*, *Hartenstein* and *Maggi* do a great deal towards strengthening the patient. *Liebe's* preparations have the advantage that no taste of the raw material remains, even when only superficially boiled, to prevent the patient wearying of it. We have already mentioned that alcohol irritates the kidneys to a great extent. It should only be given when the patient is very weak, and then only in small quantities. It is not justifiable to forbid all alcoholic drinks merely on the strength of a few experiments made on animals. Those who have been in the habit of taking them regularly before their illness will do well to continue to take small quantities. Light wine or beer is preferable to concentrated beverages; they should never be taken on an empty stomach.* Different dietetic *régime* is not necessary in cases of glomerulo-nephritis and lesions of the bacillary epithelium.

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* *Ziegler* (*Berl. Klin. Wochenschrift*, 1892, 2) prescribes milk-diet in all cases of scarlet fever. While formerly renal inflammation was very frequent after scarlet fever, during the last epidemic in the Potsdam Military Orphanage *Ziegler* did not observe any case of renal complications out of 100 patients put on milk-diet. Out of three cases where milk-diet was inapplicable nephritis occurred in two.

Dujardin-Beaumetz (*Bull. de l'Acad. de Médic.*, No. 35) remarks that for the treatment of chronic nephritis excretion of albumin is not alone of great importance, but more especially the permeability of the kidneys and the retention of toxine. The main object of treatment must be to aid the excretion of these latter and to impede their formation. For this purpose *Dujardin-Beaumetz* advises derivatives and, besides repose, intestinal antiseptics (benzo-naphthol) and, above all, "vegetarian diet" (eggs are considered harmless by *Dujardin-Beaumetz*). Where uræmia threatens he recommends absolute milk-diet, in slight cases well boiled or gelatinised meat. *Dujardin-Beaumetz* also emphasises the caloric value of certain amylaceous substances, especially of rice.—W. H. G.

After having spoken of the hygienic treatment of albuminuria and hæmaturia we must also discuss their *medication.* But the efforts which have been made in the last ten years are not at all encouraging, and there does not exist a single anti-albumin drug which one can rely on. After a short time astringents do not suit the patient, and this is especially the case with acetate of lead, which is precisely the most reliable means of stopping albumin (*Rosenstein*). Of course this drug must never be used long at a time. Tannin, especially when given as chloride of sodium or albuminate (*Lewin*, *Ribbert*), is the most easily digested; but even in such cases it exercises no influence on albumin (*Leyden*, *Wagner*, *Rosenstein*, *Briese*, *Hiller*, *Penzoldt* and others). These preparations, such as ergotin, chloride of iron (*Bohn*) and gallic acid (*Rose*), should be reserved for cases accompanied by serious hæmaturia; and still all these remedies have given us no result in several cases where chocolate-coloured sediments remain obstinately in the urine. It is even probable that the successes (few in number), which have been attributed to these remedies, were only the result of a simple coincidence. Great hopes had been laid on *fuchsin*, but the negative results noted by *Bertet*, *Budde*, *Alibert*, *Riess* and other writers soon disabused them; *Cortezao* has tried latterly to bring it up again. We must not expect any better results from benzoic acid. All these remedies injure the stomach after a time. We recommend their not being used too long. Besides, if one remembers how the quantity of albumin varies, one can easily imagine that it is not easy to calculate the effect of the different drugs. We will speak about iodide of potassium in the chapter on "Contracted Kidney".

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* We can affirm that great success certainly attends *Fürbringer's* mode of treatment. During the last few years we have treated over sixty cases according to his dietetic principles, prescribing albuminous food, together with milk and leguminous substances, and allowing the patient a moderate quantity of wine and beer if accustomed to it. As wine we prefer good old claret. The following diet-table has proved itself *mutatis mutandis* useful, laying the chief meal in the middle of the day and preferring light food for the evening: For breakfast, 7·30 A.M.: two cups of cocoa boiled in milk, or tea one-third, milk two-thirds, white bread, butter and honey, one light-boiled egg. 11 A.M.: one pint of boiled milk with a rusk. 1 P.M.: according to option, light fish (sole, whiting, trout, pike, etc.), beef, mutton, veal, chicken, mashed potatoes, light vegetables, stewed fruit, light puddings, and three times a week a plate of leguminous soup; one or two glasses of good old claret, if accustomed to wine, otherwise an alkaline mineral water. 4 P.M.: one pint of boiled milk with a dash of tea to flavour, one rusk. 7 P.M.: cold fowl, veal, soft-boiled egg, toast and butter; if accustomed, one glass of claret or one pint of beer, if not accustomed to alcohol, milk-soup. To quench thirst during the day, toast and water or alkaline mineral water in moderate quantity.—W. H. G.

(3) *Dropsy.*—General and excessive dropsy must be energetically combated against. Nevertheless, when it appears in the course of acute nephritis, it is not advisable to have recourse at once to radical means, but to wait till general treatment proves itself without result. We have often seen swollen and disfigured children have very abundant diuresis after aqua colorata, when anti-hydropic measures have failed. If dropsy is very pronounced and threatens to end fatally, one must not hesitate to attack it vigorously. One can carry it off by the sudoriparous glands, by the kidneys, by the intestines, or, finally, by mechanical means; by diaphoresis (external and internal), by diuresis, by catharsis and surgical treatment. One commences sometimes with diaphoresis, sometimes with diuresis, and if neither of these methods succeeds, the two latter are tried, either separately or together. According to our personal observations, it would seem that in whatever order one employs the drugs, the effect is the same. When there is no contra-indication we have first recourse to *external* diaphoresis, to which we often add some purgative; if this remains some time without result we make use at once of regular diuretics. As hygienic measure we recommend very hot baths, taking care that the patient is carefully wrapped up when he leaves the bath, and hot-air baths, after which the patient ought to be enveloped in very hot damp sheets; the water baths produce abundant transpiration, and the hot-air baths moderate. *Selenezki* and *Hess*, who have studied this subject latterly, have noticed a decrease of weight varying from 100 to 800 grm. after these baths. Turkish and Russian baths have also been used, but the former have the disadvantage of exposing the mouth and the nose, as well as the rest of the body, to the hot atmosphere. *Liebermeister* ordered one or two baths a day of a quarter to half an hour's duration, at a temperature of 38 to 41° C. (30 to 33° R., 100 to 105·8° F.), had the patients wrapped in blankets, and left them thus for one or two hours. By this means the quantity of perspiration secreted in adults amounted to two pounds, and in children to one. It is only in exceptional cases that the results are feeble or nil. Warm baths hardly diminish the urinary excretion; it even happens, that, in cases where the albuminuria is pronounced, the heat of the bath, while increasing the blood-pressure (*Grefberg*) and dilating the peripheric arteries (*Frey*), soothes the heart, and the urine increases at the same time as the urea is excreted; the latter originates from decomposition of the dropsical liquid which has been carried into the circulation and by the high temperature of the body. Thus one can consider the baths up to a certain point

as being capable of preventing uræmia, but, unfortunately, there are very few patients who can stand them. In spite of all possible precautions we have seen more than half of our patients obliged to give up these baths, even those which have neither pulmonary nor cardiac complications. Naturally when the patients are attacked by affections of this kind, transpiration baths are contra-indicated. In cases where one has used them the patients complain of caducity and palpitations; they felt fatigued and had congestion of the head; in fact the slightest movement was accompanied by headache and pain all over. One has tried to fight against these accidents by putting cold compresses on the head while the patient is in the bath, but the pains continued. The symptoms which follow the Turkish and Russian baths are still more dangerous and uncontrollable. The Russian baths, which do not produce any evaporation on the surface of the skin, can raise the temperature of the body to such a degree that the pulse and the respiration are increased to an alarming extent. Nevertheless, we have seen brilliant results from these baths. If it appears necessary to stop transpiration baths, one should try to replace them by wrapping the patient in cloths wetted with boiling water and well wrung out. The patient must be carefully watched. This procedure employed by *Ziemssen* has had equally good results as the baths, and one could use it where the baths could not be procured. It is a much cheaper mode of transpiration procedure, and can thus be used by a far greater number of patients. The same may be said of the concise, simple and cheap method by means of dry heat (*Benjamin*, *Nieuwstraten* and *Rosenstein*). After various trials and experiences we use in our establishment the following modifications: The lower extremities of the patient are placed under a kind of cupola constructed in the following manner. This cupola is made of iron, or rather made of bars of iron a centimetre thick. The top is covered with linen; the length of this cupola corresponds to the lower half of the body (fig. 6). Finally, the bottom consists of a plank of wood which can be slid according

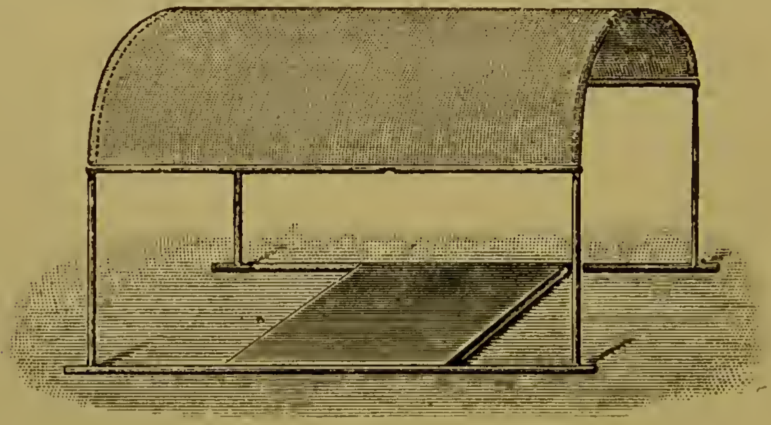

Fig. 6.

to the length of the cupola. The patient lies under this apparatus, and has between his legs a wire basket (fig. 7) which contains a spirit lamp made of metal, not of glass, to avoid the danger of an explosion. It rests with its broad basis on the wooden board, and is provided with a double cover and with a brass shade, which, when the flame is not too high, suffices to protect the linen cover. The patient can remain in this position wrapped in blankets one or two hours without being obliged to breathe too hot air, or to make the least movement; while the old system consisted of wrapping the patient in blankets and placing him under a kind of basket "figoline," and lighting a lamp under his seat; this manner was for two reasons uncomfortable for the patient: firstly, because he was obliged to leave his bed; and secondly, because he had to occupy a most uncomfortable position. Our apparatus can be used as diaphoretic means for poor patients. We must avow, contrary to *Rosenstein's* opinion, that this procedure is far from replacing transpiration baths, even if the temperature is 63°C. Many patients only transpire a little and some not at all under this cupola (it is true that one makes the same observation with transpiration baths). Nevertheless, one obtains better results with them than with hot wet cloths, and we have seen a good number of persons suffering from *Bright's* disease cured in the course of a week from severe dropsy when these baths have been used daily together with diuretics. Since using our apparatus—and we have certainly made use of it many hundreds of times—we have noticed that the flame, at first moderate, can burn up to such an extent as to cause fire. We cannot too strongly recommend that the apparatus should be provided with a handle, so that the lamp can be extinguished in an instant; if the patients are seriously ill and consequently somewhat phlegmatic we recommend their not being left alone during the procedure. We have tried our best to find some means of preventing the lamp from flaring up, but without success. A more complicated but more expensive mode of Turkish baths is *Heusner's* patent, to be had from *Pönsgen*, Düsseldorf. A winding pipe runs the length of the bed, and is heated from outside by a gas stove. We find *Quincke* and *Dubois'* apparatus much more easily portable and less expensive (to be had of *Charles Fulpius*, Geneva). The coverings are kept away from the body of the patient by means

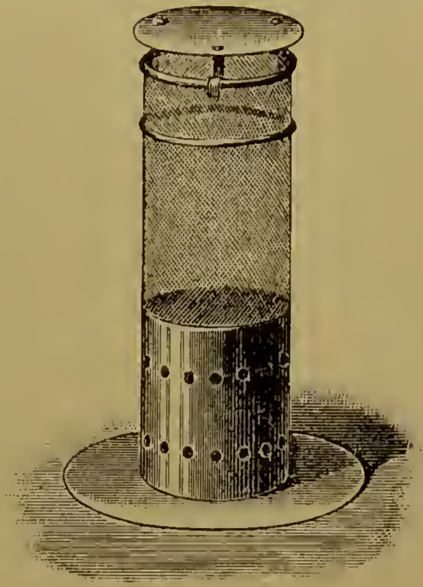
Fig. 7.

of iron rods, so that there is an empty space between the coverings and the patient. The air, heated by the lamp at the foot of the bed, is conducted into this space by means of a pipe, and that without the slightest danger of setting fire to the surroundings.

In cases of persistent anuria, which generally ends fatally, external diaphoresis does not in any way relieve the patient.

Riess declares that protracted lukewarm baths can be employed successfully against dropsy. Nevertheless, this writer, who saw the quantity of urine diminish, urges great caution in the use of them when the patient has considerable oliguria.

The best *internal* diaphoretic, which also acts as sialogogue, is *pilocarpine.* It is administered subcutaneously, 0·01 to 0·02 (these doses are reduced to one-tenth for infants), although this substance does not perhaps act with the same efficacy on the affected kidney as on the intact one. We must not forget that this drug affects the heart, weakens it, and it has been known even to cause pulmonary œdema. *Selenezki* has calculated that one dose alone of pilocarpine produces a secretion of at least 300 grammes of perspiration and 200 grammes of saliva, but these injections do not generally agree with the patient, and, like *Rosenstein* and *Henoch*, we have given up using them. We have but too often seen slight results and very great complaints. When pilocarpine produces casualties, such as collapse, painful vomiting, one must stop using it even before any decided effect is visible. If the heart be very weak or there is a pulmonary complication one must reject this remedy, which produces collapse so easily. As to believing that pilocarpine cures renal affections, as certain physicians pretend, one is simply the dupe of a strange illusion. No diaphoresis can do more, in the most favourable case, than combat against dropsy and oliguria (*cf.* "Treatment of Uræmia").

Before having recourse to derivation by means of the *intestinal tract* one must thoroughly examine the strength of the patient. Colocynth and jalap appear to be the best drugs; nevertheless, saline purgatives and senna often produce equally satisfactory results. Decided cardiac debility must contra-indicate the use of drastics.

Real diuretics were first used by *Bright* and *Christison*. One knows that excretion of urine depends on blood-pressure (filtration through the glomeruli) or on the action of the glandular epithelium (secretion); now the diuretics can act by increasing blood-pressure, or by irritation of the renal epithelium (*Munk*), or, again, by modifying the conditions of diffusibility, so that their elimination increases the quantity of water which passes through these organs. It is thus that diuretics are indicated in cases of nephritic dropsy. Certain authorities (*Ewald*, *Labadie-Lagrave*) absolutely reject the administration of all diuretics at the commencement of nephritis; but it has never been proved that they increase renal hyperæmia and affect the intact organ. *Cantani* contends that their use is illogical, inasmuch as it favours the "venous stasis," but we are completely of the same opinion as *Leyden*, *Immermann*, *Gairdner*, *Penzoldt*, *Thorn*, *Hagenbach* and others, and we think that saline diuretics are absolutely inoffensive, even when the nephritis is in full evolution. The physician of to-day will avoid the administration of squill and tincture of cantharides. *Gerhardt* and *Coignard* have rightly remarked that alkalinised urine detaches and dissolves the exudations which partially obstruct the tubuli uriniferi, and *Cantani* has verified the correctness of this statement. Studies made some years ago are said to have proved that salts of potassium influenced uræmia, whereupon we only administer the carbonate and vegetable salts of sodium; but in spite of the most careful researches we could not after all perceive that salts of potassium have had any hurtful effects, or produced uræmic disturbances. The best sort of potassium is decidedly acetate of potassium. When all diaphoretic means are exhausted we prescribe considerable doses, from 10·0 to 20·0 *pro die.* Unfortunately, this remedy frequently produces such a repugnance in the patients, that vomiting occurs, especially in women, and, as *Rosenstein* rightly observes, these coincidences become a contra-indication. This drug only exceptionally produces dyspeptic symptoms; slight diarrhœa must be considered harmless. In such cases one reduces the dose to a half or a quarter, and one mixes the remedy in a syrup or julep, which is given after meals. The effect is very rapid; if the patient is not relieved at the end of three days it is useless to continue the treatment. In cases where cardiac compensation is insufficient the principal representative of vascular diuretics, digitalis, is indicated. But where glomerulo-nephritis exists, in which the cause of oliguria is to be found in renal lesions, it is clear that digitalis, as cardiac toxic, remains without effect. In short, in spite of *Rosenstein*, we must remark, without returning to the modifications of intra-glomerular circulation of the blood, that weakness of the heart is very often the chief course of anuria and dropsy, a fact not generally believed, and we cannot recommend physicians too strongly to use digitalis carefully; this remedy has produced, according to our opinion, the best results. Our experience is but small with the Russian remedy blatta orientalis (*Bogomolow*), and we are thus unable to give an opinion as to its merits. The effect of adonis, caffein, convallamarin, strophantus and calomel has been sufficiently discussed in the part on "Engorged Kidney". We can only repeat what we said there, and affirm that tinct. strophanti possesses none of the curative properties for nephritis, which *Pins* imputes to it; we cannot warn too much against using calomel as diuretic in cases of nephritic dropsy. We know of no remedy at the same time so uncertain and so dangerous. For the large white kidney it is nearly always inefficacious, and in other cases it often shortens the patient's days.

When the various methods already described do not succeed separately one must combine them; good results have thus been obtained. Finally, when all medical means fail, one must have recourse to surgical means, but only when one has met with defeat from all therapeutic agents. Paracentesis must be made with the most minute antiseptic precaution; it does not always produce complete cure, but sometimes unexpected results. Nevertheless, we must mention that, according to our observations and experience in nephritic and especially subacute dropsy, the tendency of the serous liquids to become purulent in consequence of such an operation is much greater than in the cardiac forms. One is never sure, even with the most minute observation of every antiseptic precaution, of avoiding serious complications, such as erysipelas or gangrene, especially in private practice. Thus only turn to these double-edged manipulations as a last resource, although *Rosenstein* has recently advocated them. *Bock* has suggested a special method, he uses a capillary drainage. The punctures are made with a *Pravaz* syringe, provided with a long gutta-percha tube, which is dipped into an antiseptic. This procedure has the inconvenience of only producing a passing result and of exposing to infection. *Klaatsch's* canules are also used for this operation; they should be gilded to avoid oxidation. For the aspiration of the fluid we have successfully used a gilded canule of large calibre (1 to 2 mill.) and about eight centimetres long; this canule provided with a hard gutta-percha tube is bent and has at the end lateral openings like the drainage tubes used in surgery; one inserts them as far as the handle and changes them every two days. *Southey* and *Soulier* have recently recommended similar instruments, but the latter seems to be rather too sanguine when he pretends that all inflammatory effects are avoided by his system. Much less dangerous and more practical are the scarifications recommended by *Traube*, which we now almost exclusively use, taking care to lay a layer of turf under the patient. The scarifications can be repeated several times daily, if antiseptic precautions (bandages and cleansings) are not neglected. These scarifications produce considerable relief by freeing the affected tissues of several pounds of fluid daily. To avoid maceration of the skin we only scarify the lower part or hang the extremities in loops of antiseptic gauze. Slight hæmorrhages proceeding from the wounds are absolutely insignificant. We have repeated this treatment as often as five times in the course of a month, and have never seen the least local or general inflammatory reaction or any infection. *Straub* suggested a particular instrument for aspiration, but we consider it superfluous. If the skin bursts spontaneously even more minute antiseptic precautions must be taken.

When the patient has ascites we agree with *Ewald*, *Jessner* and *Bettelheim* in advising not to puncture too soon; firstly, not to deprive the patient too quickly of a great quantity of albumin, and then because the healing propensity of the œdematous skin becomes after a time less. We consider *Tacke's* plan of puncture with thick trocar, in order to let the ascites fluid pass into the subcutaneous connective tissue of the scrotum and of the femur and be thus resorbed, as a mistake. We have tried permanent drainage of the abdomen, which *Caillé* brought into notice, but it is very difficult to keep the canule in contact with the antiseptic liquids during the various daily functions of the patients. Fine trocars are easily obstructed, large canules should be avoided in consequence of the wounds they produce.

(4) *Uræmia.*—No complex of symptoms exists, the treatment of which has been so much discussed as that of uræmia. The one declares that he has obtained excellent results with narcotics, the second by excitants and diuretics, the third attributes the cure to aperients, the fourth to injections of pilocarpine; finally, transpiration baths and bleedings have been considered excellent, and what is the most singular is that those authors who boast of having obtained such brilliant results with their treatment abuse all other therapeutic measures. These various contradictions must not astonish us. They proceed from the fact that in some of its forms uræmia is cured spontaneously, and that the cure which one attributes to a special treatment is only a simple coincidence, and, on the other hand, that many physicians, discouraged by defeat, suffered in spite of the employment of a good method, condemn the means which they have employed; finally, one must not forget (and this is perhaps the point which especially leads to discussion) that the ensemble of the symptoms which we group together under the name uræmia are far from belonging always to the same pathogenesis. We avow that the quantity of researches which have been made on this subject latterly, as well as the multitude of opinions which have been given, have singularly modified our ideas of five years ago, in the first edition of this book. Unfortunately, our present opinions are more negative than positive. We have learnt that "uræmia" is an affection of a particular nature, which follows its course in spite of all we do against it, and which kills our patients in spite of the most "rational" treatment, and that it is cured in cases treated most "irrationally". Nevertheless, we must admit that one can attribute some success to a great number of therapeutic indications and specific remedies.

Leube, who has collected the ideas of different cliniaters, has studied the question experimentally. He draws the conclusion that when saliva, perspiration and fæces only excrete little urea one must try to produce this excretion by way of the kidneys. It would thus seem that *diuretics* should play the chief part in the treatment of uræmia. This is only partially the case. Diuresis is only indicated in certain kinds of uræmia; but it is never indicated in violent attacks or when paroxysms occur in the course of an acute nephritis. We are absolutely of *Rosenstein's* opinion, and without entering into more details on the renal lesions, we consider in these severe cases diuresis to be a very bad method. We particularly emphasise the fact that, when dropsy yields to diuretics, the uræmic affections are by no means lessened. It is particularly for that reason, and because certain uræmic appearances seem to oppose themselves to the action of diuretics, that we prefer external diaphoretic means, even in grave cases of uræmia. The treatment already described has generally shown good results. It has been said that hot baths ought to be entirely forbidden to persons suffering from uræmia; we cannot yet pronounce an opinion as to this. Uræmic disturbances have been observed as a consequence of an energetic diaphoresis, but these exceptions do not suffice to make one deny the great efficacy of baths so often seen. It is evident that baths should only be given under the superintendence of the physician. There are plenty of cases in which uræmic symptoms increase or become rapidly threatening after an extreme diaphoresis. We then immediately stop the hot baths, and we have recourse to purgatives and give a great deal of liquid. With regard to purging, if the patients are weak and the action of the heart is not too feeble, we do not even shrink from the administration of drastics (tinct. colocynth. gummi guttæ). *Lancereaux* declares that all kinds of uræmia cede to energetic purgatives; we give them, however, with great prudence and at long intervals. As beverages one can prescribe milk, alkaline waters, lemonade and weak tea; these drinks diminish the thickness of the poisoned blood in an extraordinary manner. Unfortunately, the patients are specially predisposed to vomiting and to coma, which interferes with this treatment. Enemas are auxiliaries of not great use; nevertheless, one must not consider dropsy as a contra-indication to these remedies. Finally, one has recourse to saline diuretics, especially to acetate of potassium, but this remedy produces more easily dyspeptic disturbances than it eliminates the inflammatory products of the kidney.

When all the prescribed means have been tried a great number of authorities, amongst others *Demme*, *Prætorius*, *Boegehold*, *Seemann*, have recommended subcutaneous injections of pilocarpine as anti-uræmic. We cannot approve of them without considering the various derangements already mentioned, these injections can cause the reflux of saliva in coma.

We, like *Henoch*, have never had occasion to regret having given up this remedy.

Finally, if all enumerated methods have no effect on uræmic disturbances one can have recourse to *bleeding*, which has often had brilliant results. When the pulse is strong and steady, and the patient fairly well, *Bartels*, *Ewald* and *d'Espine* recommend venesection. If the heart is weak, or in children, it is preferable to be content with cupping and putting leeches on the nape or in the temporal regions. This treatment has nothing irrational if one remembers what has been said on the theory of uræmia. It is precisely in scarlet fever uræmia that the mechanical theory has been invoked; chloroform is equally efficacious in cases of uræmic convulsions. It often stops the attack at once, probably because it acts directly on the nervous centres (*Binz*). Convulsions are often calmed by hydrate of chloral (sometimes as enema) or by subcutaneous injections of morphia. According to our personal experience, one can even prescribe them in moderate doses when there are symptoms of anæmia and weakness of the heart, especially if one is careful to use a stimulant at the same time (injections of camphor); we warn, on the other hand, strongly against ether (*Emmet*, *Hutchinson*, *von Santwoord* and others).

We willingly use *Leube's* indications. When the heart weakens day by day, as often happens in persons suffering from contracted kidney, and when it is this feebleness of heart which produces uræmic disturbances, *Leube* recommends *digitalis*, the cardiac tonic *par excellence;* if the heart is definitely weakened and there is also dropsy, he gives in addition caffein and strophantus. On the other hand, when the uræmic disturbances take place suddenly, we consider a small pulse as the surest sign of an insufficient action of the heart, and at the same time as the most decided indication for the use of camphor. We always use this drug subcutaneously, sometimes twenty doses of 1·0 gr. of officinal ol. camph. a day. In cases where the pulse is full and hard ("relative" cardiac debility) *Leube* warns against bleeding, and he prescribes digitalis; but we hasten to add that this treatment has produced several times alarming results in our patients, such as asthma, loss of strength, and somnolence, combined with oliguria. *Nothnagel* seems to have had similar experience, for he recommends the greatest caution in the use of digitalis, for, he says, when the patient passes little water digitalis can cause œdema of the brain, coma and convulsions. *Osthoff* goes still further; he declares, but without supplying us with proofs, that digitalis is dangerous, as it excites still more the vaso-motor centres, the irritation of which produces precisely augmented blood-pressure, and even causes uræmic disturbances; he believes that the beneficial action of narcotics and bleedings is only due to the lowering of the blood-pressure.

When *coma* persists we apply lukewarm baths and cold douches. According to *Labadie-Lagrave*, inhalations of oxygen after leaving the bath are good (six or eight litres of oxygen every three hours). Transfusion has been tried, but without great success.

If the patient suffers from suffocation and obstinate vomitings, he must submit to a severe diet, and be nourished artificially; in certain cases peptone may be prescribed. Of all preparations we prefer *Denaeyer's*, a clear, thin, palatable fluid which requires no preparation.* If the patient has ammoniacal vomiting, muriatic acid should be tried (*Strümpell*).

One sometimes meets with chronic forms of uræmia; we have named them abortive uræmia (see "Contracted Kidney"); these kinds generally yield to a sensible diet and abundant liquid, the utility of which has been latterly shown by *Ayer*. This author recommends the patients to pursue their usual occupations as long as possible; we cannot too strongly enforce this excellent advice. We have allowed many patients to return to their occupations, when other physicians have forbidden it, and thus gained their gratitude by allowing them to follow their accustomed pursuits.

(5) *Asthma.*—Against violent attacks of asthma sinapisms are applied. If the attacks are of uræmic origin, narcotics are to be preferred, that is to say, when the state of the heart does not interfere with the already-described treatment. *Steffen* recommends ergotin for acute dilatation of the heart. For details as to the effect of nitro-glycerine see "Contracted Kidney".

(6) *Collapse.*—Collapse is treated in every case with stimulants (subcutaneous injections of camphor, strong wines, champagne, coffee). It is evident that in these urgent cases one has not time to consider what can be hurtful to the kidneys. *Rosenbusch* has recently recommended subcutaneous injections of six per cent. solution of salt, but we have only seen very moderate results from this treatment.

(7) When the patient is attacked by *secondary inflammation* of the internal organs or dyspepsia, which, apart from uræmia, can be caused by genuine gastritis, the treatment must be regulated by the condition of the patient. If one has to combat against persistent diarrhœa (*renal dysentery*) we prescribe tannin and bilberry wine, made by *Fromm*, *Frankfort-on-the-Main.*†

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* *Brand's* essence of beef and *Wyeth's* beef juice we consider in such cases most useful preparations; we keep them in ice and administer a teaspoonful as often as the patient can take it. Double service as nutrient and as anti-emetic is rendered by milk-ice, which we prepare as follows: The white of six eggs is well beaten and added to one pint of milk. Stir the mixture and freeze it in the ice machine. A teaspoonful administered to the patient every now and then proves very beneficial.—W. H. G.

† We can also strongly recommend pastilles made of fructus myrtilli.—W. H. G.

Finally, for those patients who are not obliged to be kept in bed we recommend a season in a watering place; alkaline, alkaline-muriatic, alkaline-saline springs, as well as chalybeate waters, are particularly efficacious. According to *Senator*, the influence of drinking the waters is due to the improvement of the digestive functions, to the disburdening of the kidneys by excretion of the end products of circulation. According to our experience thermæ and hot salt springs are the most favourable. *Vichy* is highly to be recommended. *Battaglia* is also very good.* It is superfluous to remark that the patient should we warmly clad, particularly if he inhabits northern regions. Climatic health resorts, especially in warm countries, are often of great value (Riviera and Egypt).† Finally, *Senator* emphasises the importance of the patient avoiding all kinds of mental excitement; this advice is of the greatest consequence; we have often seen the detrimental effect of such psychical influences.

3. Contracted Kidney.

Under this heading we understand all kinds of diffused nephritis, which have the three following characteristics :—

(1) The elements of the kidney are contracted, and often to such an extent that this contraction is visible to the naked eye.

(2) They have one special clinical form.

(3) Their development is slow, insidious and sometimes interrupted by violent attacks.

This kind of nephritis was termed by *Bright* and *Christison* "granular atrophy, degeneration," and was considered as the "third" stage of *Bright's* disease; but these theories were disputed by *Wilks* and *Bartels*, who consider (with little justification) "small contracted kidney" or "genuine contracted kidney" as an independent process. The "rein goutteux" of the French, the "gouty kidney" of the English and *Leyden's* "renal sclerosis" only correspond to our classification, in so far as the latter understands under renal sclerosis also *Bartels'* secondary contracted kidney. On the other hand, as the majority of cases of contracted kidney are only the final product of a lingering diffused nephritis, it follows that our group is not without connection with that of chronic diffused nephritis.

From an anatomical, pathological point of view our contracted kidney corresponds with *Ewald's* "atrophied hard kidney". We shall see by the following what importance one must attribute to the numerous synonyms which are employed, such as "granular atrophy" ("granular regression," "granular degeneration"), "chronic interstitial nephritis," "nephritis atrophicans," "renal cirrhosis," "small white and red kidney". We have not cited the contracted amyloid kidney; it is of the same kind as the large amyloid nephritic kidney; the more the organ is contracted and the less decided the amyloid degeneration is, the more the nephritis resembles anatomically and clinically the contracted type.

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* Baden-Baden, Buxton, Harrogate, Montecatini, Wiesbaden.—W. H. G.

† St. Leonards, Torquay, Ventnor.—W. H. G.

Etiology and pathogenesis.—The secondary contracted kidney and a whole series of inflammatory processes of the same nature, which tend to the retraction of the kidney, have the same origin as chronic diffused nephritis. We have no other data for the causes of this affection of which the course is so insidious. It rarely attacks children (*Förster*, however, recognised it latterly in two cases post mortem); the second half of life is the most usual age for it. It is probable that ignorance is the cause of the patients only consulting us after they have had the malady for years. In a certain number of cases one accuses *chronic saturnine poisoning*, *gout*, *arterio-sclerosis* and analogous maladies of the vessels. We will examine the etiological facts one after another, because they produce at first atrophy without the renal parenchyma having to submit to a previous inflammatory process. In consequence of this the contracted kidney cannot be considered as the final result of one sole kind of affection, but of a certain number of different processes (*Leyden.*)

(1) *Saturnine poisoning.*—In spite of the opinion and the negative experiences of *Bamberger*, *Heubel*, *Jaccoud* and *Rosenstein* we consider the influence of saturnine poisoning as proved in the etiology of nephritis. Twenty years have elapsed since *Ollivier* studied experimentally the connection between saturnine poisoning and albuminuria. *Gaucher* and *Seguin* have studied it clinically; then *Charcot* and *Gombault* have taken up the question again latterly, and declared that the administration of lead to guinea pigs produces at the end of a certain time symptoms of contracted kidney, and that some months later one has found the kidneys on section in a state of granular atrophy. *Maier* and *Molenaar* have also met with interstitial and desquamative changes of the kidney. Finally, *Hoffa's* experimental researches confirm these facts. This author has not met with the typical granular kidney in guinea pigs and rabbits, but he found on the surface of the organ interstitial and tubular changes, as well as modifications of the vascular apparatus. On the other hand, *Dickinson* saw out of forty-six cases of chronic lead poisoning twenty-six times contracted kidney, and *Wagner* noted out of 150 cases of contracted kidney, which he observed in Leipzig, fifteen cases of lead poisoning (especially in painters and type setters). We could add to these some very demonstrative cases observed by *Lancereaux*, *Leyden*, *Geppert*, *Lublinski*. We ourselves have observed albuminuria in a large number of patients attacked with saturnine colic, and we have even seen in some cases lasting

symptoms of *Bright's* disease. *Rosenstein* has latterly returned to his former opinion, and he admits now that lead has an indirect connection with nephritis, but he refuses to believe that *it alone* can produce *Bright's* disease. Although there is not much unity of opinion as to the maladies which occur amongst lead miners, it is worthy of notice that out of nine patients suffering from genuine contracted kidney *Jacob* observed that eight were lead miners. One does not yet know in what manner this is produced; whether it results from an arterial affection, caused by the continual influence of the poisonous substance on the vascular walls (according to *Rossbach-Rosenstirn* the lead salts have a contracting action on the vascular lumina), or in a depositing of lead in corpuscular form (*Leyden*, *Litten*); or, finally, from the presence of a primary epithelial necrosis, resulting from fusion formed between the lead and the cellular protoplasma (*Cornil*, *Hoffa*). It is important, from an etiological point of view, to know that the contracted lead kidney, in its greatest analogy with so-called arterio-sclerotic, renal atrophy, appears in young persons where the presence of arterio-sclerosis must be denied (*Leyden*). *Dickinson* and *Lancereaux* believed that lead intoxication only produces nephritis by means of gout, and that gout is an indispensable intermediary between lead poisoning and nephritis, but these restrictions fall to the ground of themselves, because in the immense majority of contracted kidney the patients are not gouty. Amongst *Jacob's* observations (which we mentioned above) there was not one gouty patient. One must attach less importance to *Musehold* and *Lublinski's* observations, who never met with deposits of uric acid in contracted kidney. *Tancquerel des Planches* has described 752 cases of lead poisoning accompanied by arthralgia, but one knows that the identity of arthralgia and gout is not yet sufficiently established.

(2) *Gout.*—This etiology has been already conjectured by *Rayer* and *Todd;* the latter created the expression "gouty kidney," which has been confirmed by *Garrod* and *Dickinson.* In Germany one occupies oneself in general but little with this form, arising doubtless from the fact that gout is rare in that country. One must not believe that the gouty kidney is always accompanied by deposits of urates (*Charcot*, *Ebstein*); on the contrary, one has met with undeniable cases of gouty kidney without gouty deposits in the joints, such as *Ebstein's* "primary gouty kidney". The latter draws the conclusion from his experiments that the gouty substances which are not easily soluble, and which contain combinations based on uric acid, attack the renal tissues in a liquid state, and that it is only in the mortified regions that urate of sodium crystallises itself. On the other hand, *Virchow* regards the facts described by *Ebstein* as exceptional, and he thinks that the interstitial inflammation of the cortex is generally independent of the deposits of urates which one meets

with sometimes. He has proved in a most satisfactory manner that the augmentation of uric acids, even when they are still liquid, suffices to cause nephritis uratica. We have examined two gouty kidneys, one presented the alterations described by *Ebstein* and in the other there were none to be found (see "Pathological Anatomy").

(3) *Vascular changes.*—The etiological influence of vascular diseases has been recently disputed, and especially the relationship between genuine contracted kidney and arterio-sclerosis. According to *Leyden*, these two affections are not without connection, but *Wagner* and *Rosenstein* do not share this opinion, and the latter declares the arterio-sclerotic contracted kidney to be a fact of secondary importance; he distinguishes it from the genuine granular kidney, and considers the vascular changes as secondary, at least in the majority of cases of renal cirrhosis. At a certain period we allowed ourselves to be influenced by some isolated cases, and we admitted, like *Gull* and *Sutton*, that the ordinary contracted kidney began with changes in the smaller vessels. But after having pursued our clinical and anatomical studies on a large scale we returned to our former ideas, and own that, if primary arterio-sclerotic kidney exists, these cases are exceptional in comparison to those which have real chronic nephritis as their origin.

For information on *Semmola's* supposition as to the origin of contracted kidney see the part on "Etiology of Chronic Diffused Nephritis".

A certain hereditary influence is very probable (*Samelsohn*, *Eichhorst*) also in cases where gout is not intermediate.

Pathological anatomy.—We have seen that chronic diffused nephritis, when it has existed some time, can produce a contraction in the kidney visible to the naked eye. We do not call such kidneys contracted kidney, but reserve this term for the cases in which the contracted centres have gained preponderance over the normal excretive parenchyma. We have also seen that the processes of retraction are not always necessarily accompanied by a diminution of the organ. Observations are even quoted in which contracted kidney was increased in volume, and where the atrophic process was only visible under the microscope; in these organs hypertrophy of the interstitial tissue had overcompensated glandular atrophy. But these are exceptions, and in the majority of cases one finds post mortem small and contracted kidney without being able to affirm, if these kidneys have been enlarged at a certain moment, or whether the whole process has been so gradual that it never came to an increase of volume.

The classic form of contracted kidney *scil.* the bilateral one—unilateral contracted kidney has its local causes and does not belong to this description—is as follows: The organ is very much reduced in size; its diameter hardly reaches half of its usual size; it is hard, resembling hard leather, and surrounded with fat. The capsule is thickened, adhering firmly to the surface of the kidney, and on removing it, small portions of kidney substance tear away with it. The exposed surface of the cortex is uneven, covered with granulations. The depressions are pink or red, and composed of vascular retracted connective tissues. The protuberances are of a yellowish grey colour, and their parenchymatous nature is evident to the naked eye; they are composed of relatively unchanged granular tissues. According as the elements are dark coloured, bordering on maroon colour, or, on the contrary, light coloured, we have the type of the "red" or the "white" contracted kidney. Probably no essential difference exists between these two kinds, but they are parallel forms with gradual differences, caused by hyperæmia or anæmic conditions, or more or less pronounced fatty degenerations (*Weigert*, *Wagner*). We must, however, add that the small red kidney, much more uncommon, is hardly ever secondary, and represents a more advanced degree of contraction. Besides the granular masses, cysts stud the surface, varying in size from a pin's head to a pea; they contain a yellowish, transparent liquid; besides, there are small white nodules to be found (*Sabourin*).

On section it will be found that the wasting of the organ occurs chiefly in the cortical portion, it may be reduced to the thickness of a millimetre. The characteristics which we have noticed on the surface of the organ are continued in the cortex, one finds atrophic cicatricial structures, striated, cuneiform and even reticular. The pyramid portions, of rather a darker colour, are not so considerably affected, they are generally moderately contracted, while the pelvis is relatively enlarged.

Finally, the walls of the renal artery are in a number of cases thickened and its lumen contracted.

Microscopical details on the nature of contracted kidney are to be found in the part on "Chronic Diffused Nephritis". It is not uncommon that more than half of the cortex is attacked by atrophy; its centre shows itself at first in the elongation of *Bertin's* columns; the pyramids are only affected later on. These centres are formed by a connective tissue more or less vascular and infiltrated with embryo-cells, which disappear at the end of a certain time. The tubuli uriniferi occupying the place of these centres have completely disappeared, or at any rate are only represented by narrow atrophic and cloudy fibres, which have no lumen and are composed of fatty degenerated cells. The glomeruli also present various stages of atrophy, such as have been already characterised by *Frerichs* and *Beer*, later on by *Klebs*, *Rindfleisch*, *Traube*, *Thoma*, *Ziegler*, *Wagner*, *Leyden*, and others. The connective tissue round the convoluted tubules, the thickening of the capsules through proliferation similar to onion skins, finally produces small, more or less shiny impermeable fibrous knots, which, by their massing together, obliterate the parenchyma. *Thoma* has shown that obliteration of the glomeruli resulted in placing the vas afferens in the closest connection with the vas efferens or with the capillary net.

The development of atrophic centres which we have just described is probably in connection with the physiological action of the different parts of certain renal parts (*Cohnheim*). *Kelsch* believes that the part least affected, the granular, responds to the zona of the straight tubules, while the depressions correspond to the atrophic parts. *Charcot* is of *Cohnheim's* opinion, but *Weigert* has proved that these facts were without much moment, or at least that they had a very secondary importance.

In order to explain the formation of the atrophied centres one must, in the majority of cases, have recourse to primary affections of the malpighian apparatus (*Wagner*). With this the atrophy of the tubuli uriniferi goes hand in hand, while the interstitial processes, as already mentioned, are generally secondary. Contrary to the theories of *Kelsch*, *Bartels* and *Dunin*, who consider the interstitial processes as the primary ones, the "pathogenic principle" is, according to *Weigert*, the same from whatever cause the nephritis, be it toxic, infectious, from changes in the blood-composition, or from vascular maladies, causing epithelial alteration. The simplest kind of contracted kidney, dependent on circulatory disturbances, is to be found in arteriosclerosis (*Leyden*). Here the obliteration of the glomeruli is followed by atrophy of the corresponding tubuli uriniferi in consequence of the cessation of their functions (primary contracted kidney) (*Ziegler*). Complicated forms on inflammatory bases are doubtless more frequent than lingering evolution and mature *a priori* the tendency to contraction. The distinction between primary and secondary kidney, introduced by *Johnson*, *Bartels* and *Leyden*, cannot be anatomically proved. At any rate the distinctions which *Bartels* and his followers considered as sufficient in order to describe a special type of secondary contracted kidney, such as a slight atrophy of the organ, often in diameter enlarged; wide tracts of cicatricial connective tissue, separating the yellow-coloured eminences, are neither characteristic nor frequent. Finally, that the pathological process producing cicatricial tissues is often complicated by embolic infarcts, especially in such patients who suffer from heart disease. Nevertheless, we believe that *Wagner* somewhat exaggerates in considering *Bartels*' distinction as purely superfluous, all the more so as small white kidneys exist, the macroscopical and microscopical characteristics of which, especially the fatty degeneration of the parenchyma, seem to demonstrate their origin from the large white kidney. *Leyden* has recently described a special kind of scarlet fever nephritis supported with anatomical preparations, which he had the kindness to show us; we ourselves have observed two similar examples, of which we could clinically verify the acute inflammatory origin.

Among the non-atrophic, cortical centres one finds spaces, more or less large, of healthy substance, of which the tubuli uriniferi are normal or even enlarged, their epithelium is smooth and a little swollen ; this latter constitutes compensatory hypertrophy (*Köster*, *Ribbert*, and others). The enlargement of the glomeruli is concomitant, and, according to *Fortlage's* recent experimental researches, can assume double the normal diameter. The dilatation of the tubuli uriniferi should be considered as compensatory, and can be also explained by eccentric cicatricial traction (*Buhl*). The involution of the tubuli uriniferi themselves, especially in cases of " secondary " white contracted kidney, is analogous to that described in chronic diffused nephritis. One also meets with casts of considerable breadth, totally obstructing the lumina of the canaliculi. Like the tubuli uriniferi, the capsules, which lie near the surface, can expand in their turn and become the origin of small cysts, containing squamous epithelium and a yellowish colloid fluid. The whitish knots noticed by *Sabourin* are small accumulations of epithelial elements, of small adenomata. Even in the non-atrophic regions the connective stroma is generally enlarged and undergoes cellular infiltration. As to the glomerular and tubular neoplasms, as well as such which proceed from the connective cicatricial elements described by *Pisenti*, we have never heard them named by other authors.

In general, and apart from certain rare complications, the affections of the pyramids consist in striæ irradiating from the cortex (atrophic loops) and much enlarged collective tubes.

In exceptional cases the small granula are missing, subsequent to diffused non-central contraction. Such kidneys, especially when not diminished in size, appear to the naked eye as normal. Microscopical examination proves the error. According to *Lancereaux* and *Musehold* the granulations are dispersed in a very irregular manner in the lead kidney ; *Gayler* has observed in such a case that endarteritis manifests itself particularly in the glomeruli, as well as in the atrophy of the muscular cells of the media of the smaller arteries. *Coen* and *D'Ajutolo* have noticed in animals intoxicated with lead, first, degeneration of the parenchyma, then hyaline glomerulitis, and finally interstitial nephritis.

Gouty kidney merits a special description. Here, as in a certain number of cases of saturnine nephritis, one observes with the naked eye small yellowish-white points and streaks of crystallised urate of sodium. These " urate infarcts " have a predilection for the papillary portion of the kidney, although the cortical part is not spared ; one finds the deposits in the cavities of the tubuli uriniferi (*Virchow*, *Charcot*, *Cornil*, *Lancereaux*, *Wagner*), as also in the interstitial tissues (*Dickinson*, *Litten*). On examining the typical gouty kidney, *Ebstein* found as an essential fact that the urates are imbedded in a structureless homogeneous substance which at the most contains remnants of mortified tubuli uriniferi with reactive inflammation at the margin. *Ebstein* considers these processes of necrosis to be characteristic of the gouty kidney. The nutritive disturbance of the tissue is the primary crystallisation of the urates, the secondary occurrence produced by the free acid found in the necrotic deposits. We have undertaken a series of experiments for the object of supporting this theory, but this is not the place to enter into details on the subject. As already remarked (*Rosenstein* particularly mentions this) there is only one kind of evolution of the gouty contracted kidney. The concretions of uric acid which one meets with in the pelvis renalis have nothing more to do with the gouty kidney than with gout itself (*Virchow*). Persons suffering from nephritis have generally articular pains; when the patients do not suffer from this, one finds (*Virchow*) arthritic nephritis without arthritis.

It remains for us now to examine the important microscopical position of the *blood-vessels*, of which we have not yet spoken, and to determine if these vascular disturbances, especially those which attack the smallest arteries, are secondary or primary.

We remember that *Thoma*, *Ewald* and *Wagner* have remarked in the small contracted kidney hypertrophy of the walls of the renal artery and diminishing of its cavity, disturbances which *Cornil*, *Ranvier*, *Friedländer* and others place on a par with those of *endarteritis obliterans*, and which are not unconnected with the atrophical processes one observes in the domain of the interlobular vessels. This hypertrophy shows itself very soon; *Fischl* and *Schütz* have tried to constitute a group of nephritis characterised by glomerulo-nephritis and endarteritis in the absence of all other disturbances. The essential process consists in growth of the intima, without particular proliferation of the nuclei, and can completely obstruct the lumen of the vessel; the muscularis is, on the contrary, nearly intact, and is only rarely replaced by connective tissue.

One must be careful to distinguish endarteritis obliterans from *hyaline-fibroid* formations, not possessing amyloid reaction, and localised outside the muscularis, but which are to be found in the intima; they obliterate the cavities of the vessels, and, according to *Holsti*, these formations occupy the whole of the vascular walls. These vascular changes were first discovered by *Gull* and *Sutton*, and described as *arterio-capillary-fibrosis;* it is probable that this affection is not without analogy with the "hyalinosis" of modern authors. In all cases, according to *Gull* and *Sutton*, this degeneration of the small arteries and capillaries can establish itself in various organs, in the lungs, heart, brain, stomach, etc., and, according to *Lemcke*, even in the skin. When these disturbances are found in the kidneys, they produce by compression atrophy of the canaliculi and, as partial manifestation, the small granular contracted kidney. Nevertheless, this theory, which attributes the contracted kidney to primary vascular disturbance, did not remain long unattacked (*Grainger Stewart*, *Galabin*). *Johnson* even declares that vascular degeneration is only a post-mortem phenomenon, which is, however, not the case. In fact *Sotnitschewsky* first proved the existence of this degeneration in the course of development of the contracted kidney, then *Thoma*, *Mahomed*, *Leyden* and *Senator* agreed in all points with *Gull* and *Sutton's* ideas of the vascular disturbances being primary, and these authors proved a chronic fibrous endarteritis, corresponding absolutely with *Friedländer's* endarteritis obliterans, and which produces gradually analogous degeneration, as described by *Gull* and *Sutton*. When the hypertrophic process produced in the interlobular spaces generalises itself and extends into the renal parenchyma, this latter becomes atrophied at the same time as the kidney diminishes. Further, the walls of the capillaries become abnormally permeable and constitute the already-mentioned change in circulation. *Cohnheim*, however, considers in genuine granular atrophy of young individuals the thickening of the vascular walls only as *Friedländer's* secondary arteritis, which so frequently accompanies interstitial inflammations of various organs. *Buhl*, *Rindfleisch*, *Southey* and *Ewald* share *Cohnheim's* opinion. *Ewald* affirmed in nearly all the kidneys attacked by granular atrophy, muscularis hypertrophy of the arteries in different parts of the body, which *Johnson* had already named as a consequence of cardiac hypertrophy. *Rosenstein* considers this thickening of the media as the most frequent vascular affection, and insists on the rarity of the degeneration indicated by *Gull* and *Sutton*.

On the other hand, *Ziegler* pronounces himself in favour of a primary arterio-sclerosis, being the origin of a certain number of cases of contracted kidney; *Leyden* confirms *Gull* and *Sutton's* opinion in all points; *Klebs* admits a special type of English authors' "red granular kidney" (*Bartels*' "genuine contracted kidney") which is especially found in persons advanced in age, and which he considers as a non-inflammatory but degenerative process, ensuing from general arterio-sclerosis. In quite a series of cases *Leyden* observed at the same time the disturbances indicated by *Gull*, *Sutton*, *Thoma* and *Ewald's* arteritis.

Finally, *Wagner* has devoted long studies to the arterial affections of contracted kidney, and following his description in broad traces we mention the following categories: (1) Contracted kidney, presenting all the clinical and anatomical symptoms belonging to this kind of nephritis. It is found in patients of more than forty years of age, together with chronic endarteritis of the aorta and the large arteries. These arterial lesions are met with in individuals whose kidneys and urine are absolutely normal. One does not yet know if they are primary or secondary, or whether the contracted kidney has not a third unknown origin. (2) A category of contracted kidney exists in individuals of all ages (from sixteen years and upwards); this nephritis is sometimes accompanied by general sclerosis of the aorta and larger arteries, when the regions of the renal arteries, even to their smallest ramifications, are attacked by endarteritis obliterans. In such cases of renal atrophy *Wagner* only allows a primary affection of the smallest ramifications of the renal artery, when produced by syphilis. He, otherwise, considers endarteritis, which is only to be found in other renal diseases (hydronephrosis, primary renal tuberculosis, unilateral atrophy after pyelitis), as a secondary affection.

We have made a great number of researches which are, unfortunately, not yet concluded, and we cannot express ourselves definitely. Until now we are persuaded that in the majority of cases of granular atrophy (the small red kidney included), one finds the cause in a genuine lingering nephritis, and the changes in the small arteries, nearly invariably to be found in the kidney, to be nothing else but secondary *endarteritis obliterans* accompanying the process of inflammation. This often corresponds with *Johnson* and *Ewald's* hypertrophy of the muscularis. We agree for the following reasons entirely with *Wagner* and *Rosenstein*: (1) The lesions of diffused nephritis and those of the vessels are far from constant; they are sometimes very slight in cases of intense nephritis; *Thoinot* and *Darier* have seen them completely missing in some cases. (2) The thickening of the walls of the vessels is not always of a hyaline nature, as *Gull*, *Sutton* and *Leyden* have indicated. (3) In the majority of cases urinary sediments are abundant. (4) Finally, one meets with absolutely identical contracted kidney in young persons, where there has never been a question of generalised arterio-sclerosis. On the other hand, and without having a great number of proofs, we must accord to *Leyden* that, as well as chronic endarteritis, which is only secondary in the development of nephritis, arterial disturbances, degenerative and primary, exist; disturbances which come from arterio-sclerosis, and which, by reason of the diminution of the blood supply, produce little by little contracted kidney of vascular origin ("Gefäss-Schrumpfniere"). These modifications can even lead to genuine granular atrophy, the symptoms of which are similar to those of contracted kidney, and which do not admit of comparison with the moderate contractions of the cortex, frequently to be found where the capsule does not adhere, and where during life the symptoms of contracted kidney are to be found. We meet with this kind in cases of engorged kidney and in aged persons. For these reasons we consider it to be more logical to describe here and not in a special chapter these forms of vascular origin of which symptomatology is characteristic, and of which parenchymatous affections, that is to say renal necrosis, only plays a small part. But we draw attention to the fact that, according to our opinion, genuine contracted kidney, with clinical importance and extending to granular atrophy, is only exceptionally due to an arterio-sclerotic process in *Gull's* sense, and we agree with *Grawitz* that *Gull's* ideas are not in accord with renal pathology.

From a pathogenic point of view and closely connected with the red arterio-sclerotic kidney is the *senile contracted kidney*, which is based more on simple atrophy with essential proliferation of connective tissue. Nevertheless one must distinguish here also the epithelial inflammatory origin (*Ballet*). One must not confound senile contracted kidney with another form, characterised by a very weak diminution of volume, of no clinical importance, and which *Wagner* calls the *smooth senile kidney*. Apart from endarteritis obliterans of the smaller and smallest arteries, the smooth senile kidney offers no other important anatomical characters. One has even seen the trunk of the renal artery attacked by sclerotic atrophy without its being followed by a consecutive contraction (*Rosenstein*).

A complication of the greatest importance, described by *Bright*, and which one frequently meets with in the post-mortem of persons who have suffered from contracted kidney, is *hypertrophy of the left ventricle* with moderately extended or nearly normal cavity. Strong dilatation is not very frequent, but according to our experience not so seldom by far as decided concentric hypertrophy. *Senator* contends that, in general, chronic parenchymatous nephritis is accompanied by eccentric hypertrophy, while simple hypertrophy belongs more to contracted kidney; but neither we nor others have been able to verify this fact. *Spatz* has drawn the conclusion from his systematic observations, that on examining a great number of patients, one pretty frequently finds the right ventricle participating in the hypertrophy of the left one; this opinion is not without foundation. In a series of observations one has noticed myocarditic centres in the region of small obliterated coronary arteries, analogous to sclerotic renal changes; these deposits were at the same time the seat of a fatty degeneration of the cardiac muscle (*Buhl*, *Leyden*). In a similar manner *Guyot* explains his "cardiac sclerosis," and *Debove* and *Letulle* their "cœur rénal," as a partial manifestation of a general fibrous diathesis in the sense of *Gull* and *Sutton*.

As to the relationship existing between cardiac hypertrophy and renal disease, we refer our readers to the part on "Symptomatology," as also for certain special phenomena, such as dropsy, secondary inflammations, and hæmorrhages.

We omit the description of facts found post mortem in primary diseases, such as old syphilis, specific arthritic changes of the arteries, and diseases of the urinary passages.

Symptomatology.—Oliguria and dropsy, albumin and casts are, as we have seen, the principal symptoms of chronic diffused nephritis. But in contracted kidney we observe contrary symptoms; the patient suffers from *polyuria*, has but small inclination to dropsy; his urine is only *slightly albuminous*, and *poor* in *casts*. Finally, when the malady reaches an advanced stage *uræmia* completes this clinical type. These symptoms form the outline as soon as the greatest portion of the parenchyma is atrophied, be it that this atrophy is the gradual result of acute or subchronic nephritis, as characterised in the foregoing part (in these cases of "secondary" contracted kidney the albumin is the most abundant), or be it that the nephritis has taken its origin insidiously by means of highly chronic inflammation or primary vascular degeneration. As cases of the first category are much more rare, it follows that as a rule it is not possible to mark with certainty an initial stage. It is evident that while dropsy soon compels a patient suffering from symptoms of the first group to consult a physician, patients belonging to the second group remain a long time utterly unconscious of the terrible malady, which is undermining their constitution. The copiousness of their urine rarely attracts their attention, and it is only when headache, palpitation, respiratory disturbances, dyspepsia, eye and ear disturbances, paleness, neurasthenia, anæmia, emaciation, even strangury have tormented them a long time, that they consult a doctor, without the remotest idea that their sufferings are due to renal disease. Often the physician himself does not recognise the malady. Sometimes the patients succumb to an attack of apoplexy or of uræmia, or they die from an intercurrent illness, perhaps in a traumatic manner, and we find at the post-mortem an individual, who has otherwise never been ill, with completely contracted kidneys.

On turning our attention to the functional disturbances of contracted kidney, we remark that the patients pass a quantity of urine which is generally about equivalent to double, even treble the normal quantity. Quantities of ten litres and more however are exceptional cases. In our very numerous observations we have never remarked more than six litres, and it is only exceptionally that we have counted five. It is evident that intelligent and impressionable patients, who see their urine increase every day and suffer from thirst, fancy that they are suffering from diabetes. In distinction to the large white kidney the quantity of urine is tolerably in proportion to the quantity of liquid consumed (*Rosenstein*). The urine is transparent, pale; its *specific gravity* varies from 1005 to 1010, and when the quantity diminishes it remains about 1012. According to our numerous observations we accord to these characteristics of urine a great importance from a differential, diagnostic point of view; we have been able, thanks to these symptoms, to form our diagnosis more than once in doubtful cases. When the quantity of urine decreases below the normal quantity (in consequence of diarrhœa, vomiting, perspiration, fever, thirst, weakness of the heart), the specific gravity remains about the same; that is to say, that it does not vary much from the normal specific. We have even observed the specific gravity not surpassing 1018, in spite of good appetite, in a patient who passed only half a litre of urine daily. In short, the person suffering from renal cirrhosis has lost the capability of excreting the uratic salts which give urine its specific gravity. The quantity of *albumin* is either very slight or does not exist at all, generally some fraction of a gramme per cent. The absolute quantity of excreted albumin is tolerably limited in spite of polyuria. *Fleischer* mentions nine grammes of albumin *per diem* as maximum. In secondary contracted kidney we hardly noticed eight grammes *per diem*; *Rosenstein* indicates similar proportions. According to our observations the average of excreted albumin does not reach the half of these proportions. Bodily exercise increases the quantity of albumin; this augmentation is then all the more noticeable, as when the patient is reposing the albumin is hardly perceivable. It is frequently the case that on ebullition the nocturnal urine is hardly hazy, while the urine passed in the day has flaky sediment. Attacks of uræmia, inflammatory exacerbations in the kidneys and fever cause an augmentation of albumin. In some the albumin disappears for a certain time, and especially during the night; *Bartels* has observed cases where it did not reappear for weeks. *Rosenbach* watched a series of cases of transitory albuminuria which he attributed to a contracted kidney, and which appeared at the same time as violent paroxysms of headache. Finally, it has been said that albuminuria sometimes disappears for a whole year. *Schuchardt* quotes a case where the urine remained entirely normal, and albumin only appeared in the last week of the patient's life. The post-mortem showed kidneys arrived at a degree of extreme atrophy, and of which the non-atrophied parts were the seat of an inflammatory process. Especially in the first stages of contracted kidney of vascular origin albumin has a tendency to temporary disappearance. It is the same in cases in which the cardiac symptoms predominate over the renal ones (*Mahomed*, *Leyden*, *Aufrecht*). According to our own observations from analysing the urine of patients every day (which is a rather difficult task in private practice), we have seen albumin disappear for one or two days, but have very rarely remarked its disappearance for weeks or months. By testing the urine we have more than once diagnosed a renal cirrhosis in patients who have come into our hospital as suffering from heart disease, and the post-mortem has verified our diagnosis. Nevertheless one dare not be in too great a hurry to determine on contracted kidney. In cases of myocarditis, where the patients are alcoholists or suffer from hunger, the urine, the pulse and symptoms analogous to those which appear with uræmia can give the impression of contracted kidney, while in reality one finds post mortem that the kidneys are quite normal or only slightly congested. But these are exceptional cases and do not change what we have said on the subject of differential diagnosis.

Much has been said about the diminution of *urea* and the differential, diagnostic value of this symptom. *Fleischer* has made this question a subject of special study, and his opinion is that if the diminution of urea has a tendency to increase, nevertheless it would seem that it depends much on the various kinds of nourishment and the energy of the intra-organic changes. It must not astonish us that *Bartels* has observed in several cases an enormous quantity of urea (as much as 50·0 with an average of 33·0 *per diem*), and that during a long space of time. Nevertheless, we can only consider these cases as exceptional, when the kidneys are very contracted. One must not imagine that the specific gravity of urine is always in proportion to the quantity of urea (*Fleischer*). After what has already been said we need not refer again to the diminution of the uræmic excretion of urea. Usually in such cases only a few grammes *per diem* are found. An attempt has been made to establish a connection between the decrease of uric acid in urine and the presence of *arthritis uratica*, but the uric acid can diminish to a great extent in the urine without there being any trace of arthritis uratica (*Bartels*, *Fleischer*); and *Stadthagen* has recently declared that the given figures were erroneous, because they had been obtained by inexact methods of research. As to the other chemical properties of urine in persons suffering from contracted kidney, they are like others of their kind, of great theoretical interest, but they are so variable that they are without any diagnostic value. According to *Stokvis*, *Fleischer*, *Zülzer* and others, renal insufficiency is the most marked with reference to phosphoric acid.

While we always find abundant *sediment* in diffused nephritis, the urine is, on the contrary, only slightly opaque in cases of contracted kidney. Microscopical examination of the urine shows elements forming light clouds, fine hyaline casts and occasionally very broad casts; one also meets with cloudy and atrophied renal epithelial cells, and exceptionally with white and red blood corpuscles. In secondary contracted kidney elements may be observed resembling those mentioned in chronic diffused nephritis. Acute inflammatory exacerbations can give the urine the aspect of that excreted in acute nephritis,—it can even become hæmorrhagic. The presence of numerous leucocytes indicates concomitant catarrh of the urinary passages. In women microscopical examination should only be made of the urine drawn by catheter.

In spite of excessive parenchymatous atrophy, polyuria usually accompanies contracted kidney. The same is derived from the abnormal height of arterial pressure and also from the quantity of blood which flows through the kidneys in consequence of cardiac hypertrophy. The latter precisely over-compensates the decrease of urine caused by the inflammation of the parenchyma and the destruction of a certain number of glomeruli; it follows that the quantity of water which passes through the intact glomeruli is abnormally copious. *Ribbert* declares the deficient resorption of water by the medullary substance to play an unimportant part as cause of polyuria. Manifold are the reasons which have been imputed as causes of polyuria; they certainly are partly founded on false observations or erroneous theories. We have already remarked that the heart does not simply *compensate* but *over-compensates* the epithelial lesions. This fact may be explained as follows: Cardiac hypertrophy is not alone produced by destruction of the glomeruli, but also by simple inflammation and by simple congestion of the glomerular apparatus. Now this hypertrophy has less tendency of regression than the renal lesions, and furthermore, all glomeruli which undergo the reaction of a renal exacerbation are not necessarily obliterated and do not always lose their functions. Hence the over-compensating action of the heart.

On the other hand, *albuminuria* does not, as *Bartels* will have it, ensue from increased blood-pressure, but albumin and casts are produced by the inflamed portions of the contracted kidney. The minute extension of the inflammatory processes explains why albuminuria is sometimes so little pronounced and even sometimes totally absent.

As soon as the action of the hypertrophied heart becomes for some reason or other (over-exertion and inanition, asthenic effects, intercurrent febrile diseases, serious nutritive derangements, pulmonary and cardiac collapse), either temporarily or entirely insufficient, compensatory disturbances appear, which are evident by decrease of urinary excretion. The quantity of urine decreases even to below the average, and, extraordinary fact, its specific gravity only increases in weak proportion. The proportions of albumin as well as the formed elements increase. In one word, the urine of contracted kidney resembles that of chronic diffused nephritis. It is evident that in the last weeks of this disease the patient has strong decrease of urine, accompanied by dropsy and uræmia.

As already observed, cardiac compensation establishes itself in the majority of cases, be it in those of contracted kidney, be it in such of other forms. The hypertrophy is either a *simple* or *eccentric enlargement* of the *left ventricle*, and is often accompanied by dilatation of the right ventricle in cases of engorgement of various origin. Concentric hypertrophy of the left ventricle is rarely found, and occasionally only the heart is normal or even atrophied. The gradual development of the well-known clinical symptoms of hypertrophy of the left ventricle is not so easy to follow as one might suppose. *Traube* considers the heaving ictus cordis and the accentuated second aortic sound as sufficient to affirm the diagnosis; to this opinion we cannot assent, and, on the contrary, it is a mistake to reject cardiac hypertrophy when these two symptoms are missing, for the patient can be attacked by pulmonary emphysema or cardiac weakness. When hypertrophy reaches its complete development the pulse presents important peculiarities; they were so well known to *Bright* that he considered them as pathognomonic signs of renal disease. The pulse is *full*, *hard* and *of high tension*, sometimes to such an extent that it can be compared to a lead pencil or piece of wire. According to *Traube's* observations, this abnormal resistance is indisputably the sign of an augmentation of tension in the aortic system; this tension is only missing when the myocardium is entirely weakened and the quantity of blood is sensibly diminished. The distinction made by tracing the pulse curve is not so preferable as simple examination with the finger.

The troubles caused by cardiac hypertrophy announce themselves generally as initial symptoms of the malady (*Hue-Monceaux*), as in the form of headache, giddiness, tinnitus aurium, general congestion, palpitations, oppression, dyspnœa without visible affection of the lungs, genuine cardiac asthma, as also even angina pectoris. *Rosenstein's* opinion is that these disturbances always indicate an insufficient compensation; we cannot agree with this. In the first stages of the disease these disturbances are temporary; they appear in the night, or after any excess. They sometimes come and go during a year, but later, when the heart begins to weaken, they remain. Persons suffering from *Bright's* disease frequently suffer in the last weeks of the malady, like those who have cardiac disturbances, from constant dyspnœa. A *secondary* dilatation of the heart, quite apart from uræmic origin, plays a not unimportant part (*Leyden*). In other cases affections of the myocardium and fatty degeneration exist, as we have mentioned in the preceding part. *Holsti* distinguishes two stages in the evolutions of contracted kidney: a "*cardio-vascular*" period and a *uræmic* period. This distinction is often indicated, but it is not radical. According to *Bouveret*, secondary relative insufficiency of the aorta establishes itself frequently.

Gallop rhythm of the pulse, as in acute nephritis, is sometimes met with; *Potain* indicates it as a pathognomonic sign of contracted kidney, but, according to *Fräntzel*, one does not always meet with it in contracted kidney, and at other times, on the contrary, one meets with it when there is no trace of renal affection. *Johnson* believes that the not unfrequently heard reduplication of the first cardiac sound is not due to a defect of synchronism between the two ventricular contractions, but to the contraction of the auricles of the heart preceding them.

Since *Bright's* time various theories have risen up as to the relationship between cardiac hypertrophy (independent of valvular affections) and contracted kidney and nephritis in general. Even at the present time opinions vary much. We can only throw a rapid glance on the history of the question and follow in the main *Cohnheim's* clear criticism, with which we agree.

Bright's theory, that cardiac disturbances are due to the irritation produced by changes in the composition of the blood, is opposed by *Traube* with his *mechanical theory*. As on the one hand, says the latter, the contraction of the kidney and the obliteration of its capillaries prevent the blood from passing in a sufficient quantity to withdraw the water necessary for the urine, and that on the other hand the retraction acts also on the capillaries uniting the arterial to the venous system, it follows that the tension increases in the aorta. The heart, having to conquer a greater resistance, dilates itself and extends finally to hypertrophy of the left ventricle.

Traube's theory has been frequently attacked; he has been reproached, and rightly so, for attributing the augmentation of the blood-pressure to the diminution of urine and the increase of the volume of blood. In order to upset his theory it suffices to remember that one has seen considerable hypertrophies accompanying contracted kidney, which have always furnished a normal quantity of urine, and do not increase the blood-pressure any more than the most intense hydræmic plethora (*Cohnheim* and *Lichtheim*). All the same, *Rosenstein* judiciously remarks, one must not place these rapid, artificial forms of plethora on a par with slowly developed hydræmia. One must prove that serous plethora was due to oliguria, combined with increase of the quantity of water in the body, so that individuals suffering from contracted kidney absorbed more water than they excreted, and that the plethora was the primary cause of the augmentation of the blood-pressure. If one admits what has been said above, the result of this is that persons suffering from *Bright's* disease are at first polydiptics. We think that dropsical persons suffering from scarlet fever nephritis, and who take little liquid, are in a condition exactly opposite to the plethoric state.

One has gone so far as to deny the fact of renal disturbances being a primary cause of hypertrophy; and has considered these two symptoms as co-ordinate. *Gull*, *Sutton*, *Guyot*, *Edes*, *Petrone* and, to a certain extent, *Leyden* sought the general cause in the special vascular malady, to which they attributed a hyaline fibrous nature. Quite apart from the fact that this general degeneration is much more uncommon in contracted kidney than they contend, *Cohnheim* has met with cases of marked contracted kidney accompanied by cardiac hypertrophy where the large and small blood-vessels were not materially affected. But even without entering on these facts one must object to *Gull* and *Sutton's* theory, because hypertrophy can just as well exist in chronic nephritis, where there can be no question as to vascular alterations, as in senile renal sclerosis, where hypertrophy is absent in consequence of the concomitant marasmus.

There is still a fourth theory, that of *Buhl*, *Debove* and *Letulle*. The hypertrophy is, according to them, due to the modifications which take place in the heart itself; it is supposed to come from a myocarditis or from an inflammatory fatty degeneration of the cardiac muscle, that is to say, from a relative aortic stenosis which establishes itself in consequence of an alteration of the blood. This theory is not always reliable; *Cohnheim*, *Rosenstein*, *Lépine* and we ourselves have observed a whole series of contracted kidney, accompanied by cardiac hypertrophy where the myocardium was not at all changed, and of which the hypertrophy was not followed by any increase of arterial pressure.

Finally, *Longstreth* and *Da Costa*, the latter on the strength of some given anatomical, pathological discoveries, have extended their theory of the nervous origin of nephritis to cardiac complication. This latter, like the vascular changes, is the result of certain processes in the sympathetic cervical ganglions, by which the heart is innervated.

The most important point now is to explain the augmentation of blood-pressure. Followers of *Bright's* "chemical" theory have latterly tried to do this by admitting the fact of an impurity of *blood, due to the retention of the urinary products*, and especially of *urea*. According to *Ewald*, this impurity of blood prevents it passing freely into the small vessels and the capillaries. According to *Johnson*, the poisons contained in the blood irritate the muscular tunica of the arteries and cause their contraction. Be this as it may, the consequence is the same: it results in an elevation of the blood-pressure near the heart, which necessitates the greatest efforts on the part of that organ and produces finally hypertrophy of the left ventricle. This latter persists, as well as the high blood-pressure, which thus presses the necessary quantity of blood through the atrophied kidneys. As the small arteries of the organism are passively distended they would be forced to react by the contraction of their muscularis, which explains the hypertrophy of the latter already indicated by *Johnson* and *Ewald*. *Senator*, without entirely agreeing with *Gull* and *Sutton's* theory, also attributes the augmentation of the blood-pressure to overloading of the blood with urea; but we must remember that in urine of contracted kidney the proportion of urea is absolutely normal. Except in some positive observations made by *Bartels* and *Ewald*, one has never seen the blood overloaded with urea as long as the nutrition was normal and the cardiac compensation sufficient. *Fleischer* has discovered in the saliva of patients suffering from contracted kidney a certain quantity of urea without there being any uræmic disturbance or diminution of urine; that seems to prove charging of the blood with urea even in the condition of sufficient cardiac compensation. Nevertheless, the quantity of urea in the saliva is too small and inconstant, and it is so frequently totally wanting that it is difficult to attribute to it a direct influence on the nearly always constant cardiac hypertrophy. *Grützner* and *Ustimowitsch* have injected urea direct into the blood and found that the elevation of pressure which resulted from it was only of short duration. *Litten* did not succeed in producing serious disturbances in animals, even when he injected enormous quantities of urea for months at a time. It was only after having produced artificial chronic nephritis that cardiac hypertrophy was evident. Consequently it is not an established fact that hypertrophy is due to the lessened excretion of urea.

All the theories which we have mentioned up to now must be more or less accepted with caution. *Cohnheim* thought it necessary to recur to *circulatory troubles which take place in the affected kidney*, and thus he reapproaches *Traube's* hypothesis. According to *Cohnheim*, whose opinion we share, the main stress must be laid on the disproportion between the unmodified afflux of blood containing urinary substances in normal quantity, and the abnormal resistance in the vascular system behind the small renal arteries or in the glomeruli. According to *Thoma*, who relies on a certain number of personal experiments, this obstacle exists for diffused nephritis in the inflammatory disturbances of the glomeruli and capillaries which surround them, and for the contracted kidney in the disappearance of the glomeruli and in the loss of the functions of a great number of capillaries. The arterial pressure increasing in proportion as the cavity disappears, cardiac hypertrophy is the result. It is thus that it can be explained, as *Thouvenet* has recently experimentally proved, how this hypertrophy complicates double-sided hydronephrosis, how it also complicates cystic tuberculosis and cancerous kidneys and in general atrophy of both kidneys when it establishes itself in vigorous individuals. On the other hand, hypertrophy is missing when there are no circulatory disturbances, as, for example, in the amyloid kidney. Consequently *Grawitz* and *Israël* are right in saying that cardiac hypertrophy is accompanied by a certain integrity of the organism.

A certain number of authorities consider the one and the other factor, the accumulation of toxic products in the blood on the one hand, and a mechanical obstacle opposed to the course of the blood by the affected kidney on the other hand, as indispensable for the development of cardiac hypertrophy. Thus it is that *Potain* and *Hallopeau* accept *Bright* and *Cohnheim's* theory; these authors even invoke a third factor: a *reflex action* starting from the kidney and bringing with it contraction of the capillaries. But we have seen that one can only invoke intoxication of the blood in certain cases. We are not at all convinced of the fact that an intoxication is necessary for the development of hypertrophy, all the more as *Cohnheim*, while invoking an obstacle to the circulation of the blood, knew very well that the kidney was not only nourished by this liquid as all the rest of the organism, but that it is destined to receive considerable quantities of it, which are necessary to filter and to prepare; it seems to us that *Cohnheim's* adversaries have taken this fact too little into consideration.

One has experimentally tried to establish the relationship existing between diseases of the kidneys and cardiac hypertrophy; but one has not been fortunate in this matter. *Grawitz* and *Israël* have tried to atrophy a kidney and to produce hypertrophy of the left ventricle by temporary ligature of the renal artery; but although in human beings the contraction of the kidney is always accompanied by an elevation of the blood-pressure (*Basch* has seen the radial artery attain a pressure of 240 mm. of mercury instead of about 160 mm.) nothing of this kind is produced in animals. On revising his experiments *Zander* could not even find one case of cardiac hypertrophy. This latter has shown that in animals analogous conditions to human diffuse nephritis and contracted kidney could not be obtained. *Lewinski* tried to atrophy the kidneys by narrowing the renal arteries, but one must suppose that this writer has confounded a stenosis of the renal arteries with atrophy of the kidney. *Lewinski* declares he has seen a cardiac hypertrophy at the same time as a compensatory hypertrophy of the second kidney; but first, this assertion is in discord with clinical experience, and then the absence of a kidney does not in any way modify the circulation, the proof is that extirpation of a kidney does not necessitate cardiac hypertrophy (*Rosenstein*, *Simon*, *Zander*), finally, there is no retention of urea, as the healthy and hypertrophied kidney charges itself precisely with the functions which the other kidney performs. We must not forget to mention *Straus*' experiments, who, having ligated the two ureters, saw all the renal parenchyma disappear between four and six months' time; he observed cardiac hypertrophy, without the myocardium and the vessels being in the remotest degree changed.

Thirty years ago *Kirkes* and *Eulenburg* drew attention to a complication as frequent as it is serious, which accompanies cardiac hypertrophy, *viz.*, *cerebral hæmorrhage.* According to an average made from several statistics, one meets with it no less than sixteen times in 100. Out of 358 granular atrophies *Southey* has observed this complication seventy-nine times. As well as the blood-pressure there is generally a vascular disturbance (*Kirkes*, *Dickinson*). These cerebral lesions can be more or less of an apoplectic nature, they can be partial or total; their symptomatology is the same as that of cerebral hæmorrhages in general. When the patient is examined for the first time and one is not able to analyse the urine, it is sometimes very difficult to distinguish between partial paralysis and hæmiplegia. One is sometimes surprised in finding post mortem a hæmatoma of the dura mater.

The circumstances which produce cerebral hæmorrhage can also be the origin of other hæmorrhages; we will mention as examples the tenacious and rebellious epistaxis; hæmatemesis, hæmoptoë and sedes cruentae are rarer. In certain cases the clinical type resembles that of a hæmorrhagic diathesis, one even meets with hæmaturia and with urine with all hæmoglobinuric properties.

For the rest, the symptoms which we have described in chronic diffused nephritis are, apart from dropsy, similar to those met with in contracted kidney. They do not generally follow each other in the same order, their combination is more variable; they install themselves insidiously when the patient is apparently in perfect health, and then show themselves suddenly.

To these belong *eye disturbances*, in consequence of the important and characteristic *retinitis albuminurica.* They are much more frequent in cases of contracted kidney than in other forms of nephritis. On an average one meets with them in a quarter of the cases of contracted kidney, while in other forms of nephritis they do not occur more often than in one-tenth of the cases. There are a certain number of affections which one must not confound with uræmic manifestations, they can be recognised during life by ophthalmoscopic examination, and post mortem; these affections are, for example, fatty degeneration, arteritis, hæmorrhage (retinitis apoplectica), neuritis optica, choriodeal changes; they have been the subject of special study on the part of *Liebreich*, *Magnus*, *Mauthner*, *Wagner*, *Virchow*, *H. Müller*, *Schweigger*, *Hirschberg* (the latter has indicated a way of recognising arterio-capillary-fibrosis from the commencement). These affections belong to the oculist's sphere (Duke *Charles* of Bavaria has given special attention to them), and we refer our readers to works on eye diseases. As above mentioned, it is not rare that eye troubles supersede other symptoms, so much so, that it is frequently the oculist who is the first to recognise nephritis.

Furthermore, the patients are attacked by obstinate *catarrhs* of the *respiratory* and *digestive* organs, which produce painful attacks of asthma and suffocation. The digestive troubles consist of intense dyspepsia, vomitings, diarrhœa; sometimes the symptoms even resemble those of dysentery and cholera. It has happened to more than one physician that they have treated a rebellious gastric catarrh during months and years without having been able to make the etiological diagnosis, and that they have been enlightened on this point by the post-mortem. We see patients every year succumbing to "dysentery" of uræmic origin. In old age it is very difficult, even impossible, to distinguish between dysentery and uræmia. One observes the most varied uræmic symptoms, which turn out to be more or less serious derangements of the *nervous system:* giddiness, headache, especially unilateral, which *Bartels* and *Rosenstein* have often met with, as the sole morbid manifestation, great irritability, general weakness; the patients suffer sometimes from such a distaste to life that they try to commit suicide. Finally, the patients are sometimes deprived of sleep by intolerable itching of the skin and burning sensation, by neuralgia which appears under the type of sciatica with inclination to symmetrical localisation, very often also by trigeminus neuralgia, or other pains of which the localisation and the intensity are the most variable (*Berger* and the author). It is an interesting fact that the pains never have their seat in the vicinity of the kidneys; this is the "rheumatism" of inexperienced and non-conscientious physicians. Finally, we would mention the dangerous, insidious secondary *inflammation of internal organs*, specially lobular pneumonia (*Wagner* has observed twenty cases out of 150, and we ourselves have noticed relatively the same proportion), pleurisy, according to our experience not unfrequently purulent and bilateral, and pericarditis, with and without fever, but seldom with feverish urine (*Rosenstein*), rarely peritonitis. All these inflammations are often very difficult to diagnose, partly because the objective examination is impossible by reason of the deciduous state of the patient, and partly because of cutaneous œdema. In certain cases they are considered, during life, as primary, and it is only post mortem that one discovers that they are secondary. A patient in *Friedreich's* hospital suffering from decided atrophy of the kidneys complained one day of pains and of loss of appetite. An examination of the heart showed a *pericardial exudation*, to which he succumbed the next day; cases of this kind have repeatedly occurred in our hospital. One asks oneself whether endocarditis is ever a result of contracted kidney; we have had three times occasion to follow the development of this affection in an old nephritis, once even the post-mortem showed that the contracted kidney was secondary; but, in spite of these three affirmations, we think it doubtful whether endocarditis is a consequence of nephritis. Liver and spleen disturbances have nothing characteristic. In a certain number of cases one has observed the vaso-motor disturbances of the skin, and even observed symmetrical gangrene of hands and feet (*Roques*).

When the digestive organs are not attacked, and cardiac compensation is complete, *nutrition* can be perfect for years. Patients frequently even become fat (*Wagner*, *Rosenbach*). When, on the contrary, the malady becomes inveterate and intense, dyspepsia manifests itself, when the compensatory hypertrophy becomes insufficient and the troubles increase, then the nutritive functions are much affected. The patients become thinner, their skin becomes dry and pale and withered, the hair becomes hard, rigid, the features drawn and the face fallen in. Blood and fat contract like the kidney (*Biermer*). The temperature of the body sinks below the average. The facultas virilis becomes extinct. Patients suffering from renal cirrhosis are often supposed to have tuberculosis or cancer.

As we have already had occasion frequently to observe, nervous respiratory and digestive disturbances can originate from a *uræmic intoxication* which concords precisely with the retraction of the kidneys. Uræmia, in the advanced stages of the malady, when cardiac insufficiency has been for some time remarkable and the patient is weakened, usually proves fatal. The partial or total convulsions which one meets with, sometimes before all other symptoms, and which have been taken for genuine epilepsy, are less frequent than symptoms of chronic uræmia. This latter announces itself often under the form of obstinate vomitings, digestive troubles, asthma, even more frequently under the form of cardiac or bronchial affections (*Rosenstein*), and uræmia can then be mistaken for a malady of the stomach or lungs. But when these disturbances are accompanied by periodical convulsions, when the comatose patient is bathed in sticky and uric perspiration, finally, when a strong smell of urine exists, the diagnosis is evident, even for the inexperienced physician. In the majority of cases uræmia commences with weakening of the pulse, but in certain cases, on the contrary, the pulse remains normal and becomes even a little contracted. But it must be understood that these cases are exceptional, and that uræmia generally coincides with weakening of the hypertrophied heart, and with the decrease of urine resulting from it. Nevertheless, a small number of cases exists in which diminution of tension of the pulse can be missing, and, in spite of the decrease of the diuresis, augmentation of tension can be perceived.

Dropsy is no necessary adjunct of contracted kidney. The majority of patients are not attacked by it. When dropsy appears in patients suffering from contracted kidney cardiac hypertrophy has been already for some time incapable of compensating the elevation of the blood-pressure and the increased rapidity of the blood, which is produced in the parts of the kidney which still do their functions. This fact serves as explanation, why dropsy only occurs in the latter period of the illness, and that it coincides with the relaxation of the heart, which develops into a congestive condition of the whole organism.

Except in these cases which are only diagnosed at the moment, when the insufficiency of the cardiac compensation enlightens the physician, the development of contracted kidney lasts for years. According to recent observations made by *Bartels*, *Buhl* and *Wagner*, the malady frequently lasts ten years. We ourselves have seen a certain number of cases of this kind. *Biermer* and others have divided the period of evolution of contracted kidney into three stages: firstly, albuminuric stage; secondly, stage of compensatory hypertrophy; and thirdly, that of compensatory derangements, uræmia and cachexia. This division is not very exact, but it suffices as a general scheme. The principal causes of death in uræmia are secondary disturbances of internal organs and cerebral hæmorrhage. We do not need to add that death can be occasioned by an old or inter-current malady, such as cancer or tuberculosis.

The symptomatology of *senile* contracted kidney is far from being the same as that which we have just described. The urine, as *Sadler* and *Ballet* have rightly remarked, does not increase in the senile kidney, but, on the contrary, diminishes; it decreases even to a litre or half a litre. This is explained by the feeble arterial tension and the absence of cardiac hypertrophy. Albuminuria is uncommon, and uræmia seems never to occur; in a word, the senile kidney, especially the smooth one, is without clinical importance, which, however, does not prevent, as *Meigs* said, that it is more often the cause of death than is generally believed. According to a series of observations, there exists a certain number of intermediary forms between the senile and the contracted kidney.

Diagnosis.—"The physician who makes it a rule," says *Bartels*, "to consider each symptom in particular and to examine the urine of his patients when they have the slightest pain, is in a position to recognise this singular nephritis from the commencement;" and further on the same author adds: "The methodical examination of urine continued during some time and the constant observation of the heart ought to form the basis of diagnosis". These words, written many years ago, are of the same value to-day. To convince oneself of this fact, it suffices to re-read the foregoing chapter. One frequently examines the urine one single time, and if it does not contain albumin on that day one rejects the diagnosis of contracted kidney; this is a deplorable proceeding. One must watch carefully for days and months the exact quantity, the specific gravity of urine and the quantity of albumin which it contains. As to the special chemical analysis, it has not yet proved itself to be of any practical value.

Easy as it is to make the diagnosis when the urine presents the characteristic signs of contracted kidney, when the heart, the pulse, the retina and the nervous system show the changes which belong specially to nephritis, even so difficult is it to diagnose rightly when hypertrophy of the heart is not much developed, and consequently the urine is nearly normal, or even when the cardiac compensation is already disturbed. In the first case the small quantity of albumin makes one hesitate between a simple physiological albuminuria and a "latent" contracted kidney; in the second case it is the want of polyuria and the ensemble of cardiac symptoms which can lead to a false diagnosis.

It is always suspicious when albuminuria coincides with headache or dyspepsia at the same time that the pulse is contracted.

The etiology, except when it can be imputed to lead poisoning, to gout, or syphilis, is not of great importance.

To confound contracted kidney with the period of convalescence from acute nephritis is only admissible in books. The anamnesis generally indicates the right diagnosis. Polyuria can bring the idea of *diabetes insipidus*, but scrupulous observation of the rules which we have already discussed, examination of the heart and pulse, prevent an error occurring. It is less easy to distinguish *hysterical* polyuria, especially on seeing the patient for the first time. Our experience that the urine of persons suffering from contracted kidney keeps its low specific gravity, in spite of the decrease of the daily quantity below the average, is of great use.

Too much importance must not be attached to the age of the patient, it is known that even young people suffer from renal cirrhosis.

We cannot enforce the fact enough that patients who have contracted kidney can remain days and months without albuminuria; one must not, therefore, content oneself with rejecting the diagnosis of contracted kidney because of the absence of albumin and sediments.

For the reasons already given, we decline to establish in the generality of cases the differential diagnosis between *primary* and *secondary* contracted kidney, enticing as the descriptions given by various authors are. One only rarely observes sub-acute diffused nephritis existing for a certain time with all its symptoms, and gradually making way for contracted kidney. Nevertheless, we have known four cases, verified by autopsy. Besides, we have already seen that the differentiation of the two kinds has no practical importance.

Each time that one finds oneself in the presence of a cachexia when tubercles or cancer are not recognisable, one must be led to test for contracted kidney.

For details on the differential diagnosis of *amyloid* kidney, see part on this subject.

Finally, we would warn against making a precise diagnosis when one is called to a patient who is near the end of his days, and where one finds a heart deprived of energetic contraction, a feeble, irregular and frequent pulse, and when cardiac hypertrophy has disappeared. On examining a patient of this kind one can consider oneself very fortunate if an affection of the heart of a renal origin can be diagnosed. A dying patient suffering from dropsy and the most painful cardiac dysentery was brought to us one day; the symptoms led us to believe in a myocarditis with febrile endocarditis. Having found the febrile urine not to be very plentiful and containing a great deal of albumin we diagnosed nephritis. The patient died, and, at the post-mortem made a few days later, we found a contracted kidney, of which the cortex was much shrivelled up, was pale yellow, and had most of the cardiac alterations which we have mentioned. Was the endocarditis a result of the nephritis or accidental? That is a question which must remain unanswered.

Prognosis.—The parts of the kidney which are atrophied never return to their normal condition; in this sense the contracted kidney is incurable. All the more is this the case, when the malady has arrived at a period enough advanced for it to be possible to affirm the diagnosis; it always ends fatally. But a small consolation in this malady is that it can last many years without the patient suffering. Patients of this kind are not, on the whole, more to be pitied than individuals who, after having always been in good health, are carried off in their old age by pneumonia or cerebral hæmorrhage. Finally, we have seen a good number of patients who have had albuminuria and an elevation of the arterial tension lose their albuminuria and return to us years after with normal urine, which remains so for months and years. Up to which point do these cases belong to the category of simple albuminuria or to contracted kidney? Were these periods of amelioration which we have stated simple remissions or cures? These are questions which we cannot answer. What we can affirm (like several other writers) is that after having observed all symptoms of nephritis, we have noticed long remissions, one can even say that from the patient's point of view these periods were veritable intermittencies. Perhaps the intervals of health correspond to an arrest of the process of retraction.

It is true that death, as a rule, is immediately preceded by feebleness of the heart, oliguria and uræmic accidents. Nevertheless, we have seen enough patients suffering from chronic uræmia who had more or less long remissions, we have even seen persons supposed to be dying quite unexpectedly take a turn for the better and leave the hospital.

Treatment.—One can rarely treat the *indicatio causalis* of the contracted kidney, be it not lead poisoning, gout or syphilis.

The treatment of the malady itself is still more hopeless. We know of no specific drug capable of arresting the process of contraction. *Bartels* had indicated iodide of potassium, but, according to the numerous attempts, we affirm with *Cantani* and others, that this drug is without effect each time that the nephritis is not of a syphilitic origin. We insist all the more on this fact as we have several times seen oculists prescribe iodide of potassium in large doses and for a length of time with the intention of arresting nephritis, which they had discovered with the ophthalmoscope; we repeat that an anti-nephritic medicine does not exist, and that it is not rare to see dyspepsia and headache result from the use of iodide. Even there, where the syphilitic origin is certain, iodide is often useless. We have met with a certain number of cases of the same kind as those mentioned by *Mauriac*, and in which this author has praised the effect of iodide; but we must remark that the cases thus cured were more renal gummata than genuine nephritis.

Thus the physician is limited to the *indicatio symptomatica*, which, however, offers a large field for alleviating his patient's sufferings.

If good compensation exists one should content oneself with combating the congestion of the head with applications of ice, with purgatives and, with strong patients, with small bleedings. The small quantity of albumin which the patient loses is of no importance. When the patient is on the point of succumbing to apoplexy *Cantani* recommends venesection. It would be a theoretic as well as a practical error to try to combat polyuria with astringents and ergotin.

But if the compensation is disturbed the dangers are much greater. If diuresis diminishes, and the other symptoms which accompany the atony of the heart appear, one must give tonics and try digitalis. When this does not alleviate the painful palpitations one must have recourse to narcotics and local soothing remedies. If the cardiac insufficiency increases one should not hesitate to employ all the remedies we have mentioned under engorged kidney. *Smith* recommends nitrous amylether. In a certain number of cases we have seen good effects produced by a combination of morphia and cocain, a third of the latter in proportion to the morphia.

We cannot give general rules for the treatment of *dyspepsia.* The diet should be as light and strengthening as possible. It must not be forgotten that the patients have little appetite and that one must reckon with their caprices, and, above all, not insist on their eating things to which they have a dislike. Many a patient has been grateful to us for allowing him to eat what he fancied! Dyspepsia is often soothed by warm baths. Great precaution must be used in prescribing cold baths, as they increase the blood-pressure (*L. Lehmann*). Vomiting is very obstinate when it is not of uræmic origin, in which cases the indicated treatment has shown good results. Except ice, morphia and diet for several days we know no certain remedy. Tincture of iodine, creosote, nux vomica have been suggested, but we are decidedly against them. How *Rosenstein* can recommend oxalas cerei we cannot understand.

"Dysentery" in persons suffering from nephritis is more difficult to conquer than vomiting. Milk, as *Rosenstein* says, does not generally suit the patients; a suitable diet must be settled on and opium given also in the form of suppositories. Injections of water with boric acid produce good results. But generally, whether the diarrhœa is uræmic or non-uræmic, these means are of no effect.

Preparations of iron have been used against *anæmia*, but a great number of patients cannot take it, and the best plan seems to be for the patients to use a chalybeate spring. Otherwise, peptonates are to be recommended.

Neuralgic pains and migraine are calmed by antipyrin and antifebrin. Nevertheless, neuralgia of all kinds is so tenacious in the course of nephritis that the intensity of the pain alone has caused us to make a diagnosis of kidney disease.

Conservative treatment is much more efficacious than medicines. It is useless to compel the patient to perpetual rest, for this *régime* bores him more than it aids him; but all physical and mental over-exertion should be avoided; life should be made as easy as possible, all disagreeables should be kept out of the way and the patient morally supported. *Bartels* absolutely forbids his patients to drink alcohol, coffee and tea; we believe that excess alone is hurtful, and that the physician must be guided by the individuality of each patient. *Bartels* declares that the first thing to be done is to order the patient to give up all his habitual occupations; how many patients are in a position to follow this order? We do not hesitate, as *Ayer* also recommends, to let our patients follow their usual occupations, even when they are suffering from lingering uræmia.

Our remarks on the treatment of diffused nephritis hold good here, and we do not find it necessary to add anything to them. If the patients are well off they should pass the summer in the forest at a moderate height, but not too high; sea baths, especially in the south, are to be recommended. Warm clothing is of the greatest importance. According to the kind of the malady and the accompanying symptoms (obesity, gout, anæmia, heart disease), one chooses between Baden-Baden, Elster, Ems, Franzensbad, Gastein, Karlsbad, Kissingen, Marienbad, Nauheim, Pyrmont, Teplitz, Wiesbaden, etc.* The baths are frequently of great use, but the chief point is, especially when cardiac compensation is suffering, that the patient is away from all the cares of everyday life. Contracted kidney and gouty kidney cannot be put off with half a cure (*Ebstein*).

We have already entered into details as to the remedies which are used against uræmia. The most important point is to increase the energy of the heart, especially when the compensation is insufficient. *Robson* and *Bartholow* employed nitro-glycerine to diminish the arterial tension; this remedy has found an ardent advocate in *Rossbach*. We have employed this remedy in about a dozen cases, in some of which the disturbances were certainly caused by an increase of arterial pressure; in spite of this treatment we have been unable to combat uræmic asthma. In all cases we have never seen this drug diminish the tension of the pulse or increase the quantity of urine, and we do not see how it can modify the relationship between the polyuria of renal cirrhosis and cardiac hypertrophy. *Kinnicut's* statement of only having observed cases of uræmia when accompanied by great tension of the pulse, and never with feebleness of the heart, is of no great importance. *Leyden* insists, as we do, that nitroglycerine is useless in cases with asthma. Nitrite of sodium has been suggested, but we have not used it.

Inflammation of internal organs demands special treatment. We can only repeat the advice given above: to act expectatively and conservatively, and especially not to forget the poor patient through thinking too much of the remedy.

4. Amyloid Degeneration of the Kidney (Waxy or Lardaceous Degeneration).

Under this heading we include all renal affections where the vessels of the organs are the seat of amyloid degeneration, be it that the other constituents of the tissue are intact, be it that they are inflamed; the latter is generally the case, and justifies the classification of the malady under the heading nephritis. We have already seen that the more inflammation prevails and degeneration is less apparent, the more the malady approaches anatomically and clinically chronic nephritis. The affection of which we will now speak is not rare; it is, at any rate, as frequent as genuine chronic nephritis and contracted kidney together. Old statistics are not in accord with our present ideas, because not enough attention was paid to amyloid kidney. As we have seen, there is a whole series of large white kidneys which belong to this group. It has recently been enlarged at the expense of chronic *Bright's* disease, in so far as one has been contented with a trace of amyloid to include in this chapter the kidney attacked by it, to an even greater extent than much more acute parenchymatous affections, which belong more to diffused nephritis or to the contracted kidney.

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* Buxton, Harrogate, St. Leonards, Torquay, Ventnor.—W. H. G.

Forty years ago *Rokitansky* was the first who described the fatty kidney as a special form of *Bright's* disease. *Meckel*, the discoverer of the iodine-sulphuric reaction, believes in deposits of cholesterin, but this opinion has been disputed by *Virchow*, *Kekulé*, *Friedreich*; *Kühne* and other authorities show that the amyloid substance was not formed by carbohydrates, but is formed by albuminoid bodies.

Up to the present time one has not been able to settle precisely the relationship between amyloid and hyaline degeneration (*v. Recklinghausen*). In the first edition of this work we implied that the processes were very similar, and that one could consider hyaline degeneration as a *first degree of the amyloid degeneration*; the researches which we have since made in medical literature confirm this opinion. It seems that the farther we go, the more the writers are inclined to consider these two substances as two forms of one single process, and they only see in hyaline substance a preparatory degree to amyloid substance. This latter is said to have the same physical and chemical properties as the first (transparency, homogeneity, insolubility in alkali, acids, etc.), but should give a special reaction with iodine and sulphuric acid, as well as with aniline colours. All the same, these two modifications of the tissues are often found one beside the other, and it is very difficult to establish precise limit between them.

Etiology.—Primary amyloid degeneration of the kidneys is very exceptional. This malady usually appears as manifestation of a *constitutional* disease, especially of diathesis, which leads to chronic cachexia. It is most frequent in persons from twenty to fifty years of age (*Fehr*, *Hennings*, etc.).

Patients who have been suffering for a long time from multiple suppuration of the flesh and bones are predisposed for this malady. We have seen these latter affections play a very important part in the etiology of chronic diffused nephritis.

According to *Wagner's* statistics, who saw 265 cases verified by post-mortem, amyloid degeneration is most frequently produced by *pulmonary tuberculosis* (ulcerous), often accompanied by intestinal tuberculosis. *Bartels* has noticed that degeneration of the kidney was nearly always a consequence of the formation of a cavern; he concludes from this that the contact of the cavities with the atmosphere is essential to this malady, as the latter could not be produced without the action of certain substances contained in the air. But we have had occasion to verify *Bartels'* assertion, and have found that this rule has many exceptions. We have, besides, seen more than once purulent centres in constant communication with the atmosphere without there being the least amyloid process. One very remarkable fact has been noticed by *Meckel*, and that is, that the development of amyloid degeneration often coincides with an arrest of pulmonary tuberculosis, and that this latter even seems to recede.

Next to tuberculosis, *chronic affections of the bones and arthropyosis* most usually accompany amyloid degeneration. Scrofulous caries and necrosis, spondylarthrocace, coxalgia and wounds from gunshot play an important part (*Cohnheim*).

Then comes *syphilis*, especially the inveterate and hereditary forms. It is sometimes unaccompanied by any ulceration or cachexia.

Among the rarer causes are ulcus cruris (*H. Fischer*), empyema, bronchiectasis, so-called chronic dysentery, malaria cachexia (*Axel Key*, *Rosenstein*, *Malmsten*), chronic peritonitis, puerperal and other pelvic suppurations of the most varied kinds, ulcerating cancer, especially of the womb, abscesses in the kidney, pyelitis, etc. Abscess of one kidney can infect the other. We have constantly seen with suppuration of *one* kidney, that *both* kidneys were suffering from diffused nephritis and amyloid degeneration in the highest degree; but, nevertheless, we do not believe that chronic nephritis or contracted kidney can produce amyloid kidney.

Bouchard and *Charrin* have latterly produced amyloid kidney experimentally in rabbits by means of repeated inoculations of blue-pus tubercle-bacilli.

In general, it is not only the kidneys which suffer from amyloid degeneration; the supra-renal capsule, the spleen, and the liver share it (we do not know if it is true that these organs are generally attacked first). Finally, it is exceptional that the kidneys are intact when the amyloid degeneration is of at all ancient date.

The exact pathogenesis, that is to say the mechanism which causes amyloid deposits, is still obscure. It has been said that amyloid substances were in the blood, but that has not been proved.

Pathological anatomy.—From the *macroscopical* point of view the amyloid kidney shows itself in three principal forms, besides many intermediary ones:—

(1) The kidney is completely normal to the naked eye; only the chemical reactions and the microscope reveal its lesions.

(2) The organ is nearly twice its usual size; the capsule is fine and easily removed. The surface of the organ is smooth and of a pale yellow colour; on section the cortex is very thick, hard and friable, cirrhoid, and its colour is pale yellow all over. On nearer observation one sees it to be covered with spots of a greyish red colour, without hæmorrhage; and in a certain number of

cases the glomeruli appear like greyish white dewdrops, more or less sparkling (*Meckel*). The dark red colour of the pyramids contrasts with the paleness of the cortex; sometimes. however, these pyramids are pale. This form is the *large white amyloid kidney*, the "butter kidney"; doubtless the most frequent form of the malady, and that which formerly was ranged amongst *Weigert's* "chronic parenchymatous nephritis"; in fact may be considered so when the process recedes.

(3) The kidney has the appearance of an ordinary contracted kidney; it can only be distinguished by more or less marked retraction (sometimes even, when the amyloid kidney is of syphilitic origin, the kidney is a little increased in volume); on the surface a little more sparkling, and the organ a little paler than in the contracted kidney. This form is the lardaceous contracted kidney, and is peculiar to syphilis of slow evolution, but is much more uncommon than the afore-mentioned kind.*

Between these three forms one observes an infinite variety of kinds, and transitional connective forms are not wanting.†

Under the *microscope* one sees two kinds of lesions: on the one hand, those which are characteristic of amyloid degeneration, and, on the other hand, those belonging to the regular tissues of the kidneys. The latter are not found in the first-mentioned group; in other words, *pure* amyloid kidneys are found. *Cornil* and *Ranvier* have made an error in denying this.

What first strikes us in amyloid process is the inconstancy of its localisation. It is in fact most variable, and it is only as a rule, with a *very* large margin, that one can say that the process attacks by preference *glomeruli* by developing their loops and tufts and *afferent* vessels. The vasa efferentia of the cortex, and the vasa recta are less attacked. In certain cases the process attacks the medullary substance, which soon becomes pale. In the vessels themselves it is sometimes the membrana propria, especially that of the straight canaliculi (*Eberth*), sometimes the epithelium, especially at the apex of the cones (*Kyber*), or the hard hyaline cylinders, which can be the seat of degeneration. *Kyber* found in the interior of the canaliculi amorphous crumbled masses, apparently originating from the epithelium; they showed amyloid reaction. *Bartels* has met with similar disseminated elements in different parts of the kidney, and he describes them as sedimentary matter. Be that as it may, the size of the organ does not depend on the amyloid degeneration, but on the lesions of the other renal tissues. For precise histological details on the subject of the shiny thickening of the arterial walls (tunica media, intima) and the rest of the renal lesions, we refer our readers to the chapters on this subject in the manuals of instruction on pathological anatomy. It was formerly believed that the amyloid vessels were obstructed, but about twenty years ago *Munzel* made an injection into these vessels post mortem. *Litten* has recently made the same experiment.

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* In a case observed by *Grainger Stewart* the kidneys were found after death to weigh two and a half ounces each.—W. H. G.

† Three stages of the disease are also recognised by *Grainger Stewart:* in the first stage the vessels only being affected; in the second the renal tissues being infiltrated with the albuminoid material and a great number of the tubules choked with it; and in the third the organ being atrophied and contracted.—W. H. G.

When the amyloid is very marked, diagnosis is possible with the naked eye; it suffices to wash the blood away from the surface of the organ as much as possible, and then to use a solution of iodine. The points which are the seat of a fatty degeneration are red. If one adds sulphuric acid the reddish-brown colour changes into a blue-violet; this new reaction, which *Virchow* considers as characteristic of amyloid, does not take place when the kidney is only in the preparatory stage of amyloid degeneration; that is to say, the hyaline period. In order to make microscopical preparations one must lay the sections into *Lugol's* solution, then let sulphuric acid beneath the cover-glass; the amyloid parts appear then, in the majority of cases, to be of a brown-violet colour, verging on blue, and in other cases they appear to be of a beautiful blue. Sometimes the sulphuric acid hardly modifies the colour produced by iodine; in these cases the degeneration is not of a very decided nature, and varies between so-called hyaline and amyloid degeneration; some years ago we noticed that aniline gave in such cases very clear reactions. Amongst these reagents the principal ones are methylene and gentian violet; but they are much less sure than the former, for they can remain without action, while iodine and sulphuric acid always react. Aniline colours tinge the amyloid centres bright red on a blue-violet ground. Methylene green (which contains methylene violet) produces even more striking contrasts. In fact the reaction of the aniline colours is more sensible than that of iodine or sulphuric acid. Besides, if one prepares the surface of section of the kidney with aniline colours, and if the amyloid is intense, the organ presents to the naked eye beautiful designs; if one discolours the kidney a little with vinegar or weak alcohol, the glomeruli appear to be bright red; they resemble drops of sealing-wax.

When one finds oneself in the presence of the *large white amyloid kidney*, the microscope reveals, besides the degenerative processes, characteristic disturbances of the *fatty kidney*, or of *chronic diffused nephritis;* one sees the epithelial cells in all stages, from cloudiness to fatty degeneration, and at the same time one can see glomerulo-nephritis and interstitial infiltration, and even limited centres of contraction. The casts have the characters as already mentioned on the subject of the urinary deposits.

Wagner found these casts in such a number that he attributed the hypertrophy of the organ to their presence.

In the fatty *contracted kidney* one meets at the same time as the process of amyloid degeneration processes of intense contraction, which we have fully discussed in the preceding part. The atrophied glomeruli generally produce amyloid reaction.

The most experienced authors consider the alterations of the connective tissue as a sequel to amyloid degeneration; and the fact that the obstacle to opposing the course of the blood produced by the latter retracts the cavity of the vessels thus renders this opinion very plausible. Nevertheless, we must remark, regarding not only amyloid degeneration, but, more properly speaking, inflammatory processes, that nothing noticeable appears in the other organs, such as the liver, heart, lymphatic glands, and one is tempted to ask oneself, if *Cohnheim* is not right in considering amyloid kidney and nephritis as two *independent processes.* The fact frequently observed by us that nephritis is highly developed under the form of spotted, speckled, large white and atrophied kidney, while amyloid degeneration is only in the first stage, speaks for this opinion. It is easy to explain the frequency of the coincidence of these affections by the fact that they have a common origin. Amyloid can follow or precede nephritis (*Klebs*, *Ebstein*, *Litten*, *Leyden* and others). The opinion that nephritis is primary and amyloid degeneration a sequel, is refuted by the cases in which one finds nephritis without degeneration.

For dropsy, cardiac hypertrophy and other concomitant manifestations of the amyloid kidney, compare the symptomatology.

The description of the amyloid of the other organs and of their anatomical changes should be made with the principal malady (phthisis, syphilis, caries, etc.) and does not belong here.

Symptomatology.—If one remembers:—

(1) That the chief malady often connected with cachexia follows its course at the same time as the amyloid kidney;

(2) That, besides the kidney, other important organs almost always take part in amyloid degeneration;

(3) That fatty degeneration usually accompanies nephritis or contraction of the kidney, it is easily imagined how difficult it is to describe a clinical type which corresponds at the same time with all the symptoms which amyloid kidney can present, and with all the anatomical lesions by which it can be attacked. The urine is exceedingly variable; neither albumin nor urea is in steady proportions, and the daily quantity of urine passed and its specific gravity are not easily determined. It even happens in some cases that the urine presents symptoms entirely opposed to those which it presents in others, so that there is not one case in which the examination of urine can furnish pathognomonic symptoms. Anæmia, cachexia, dropsy, diarrhœa, fever, are some-

times entirely in disaccord with what happens on the part of urinary excretion.* This may explain how *Traube* describes a normal diuresis or one a little augmented with decrease of the specific gravity at the commencement of the malady, and at a more advanced period one with fever and congestion and less plentiful urine, red and of increased density. *Bartels* and *Rosenstein* have seen normal urine in cases of amyloid kidney, and finally, *Wagner* has observed oliguria in most cases.

Nevertheless, we will try to establish the ensemble of the characteristic symptoms which the testing of urine can furnish, and .which respond as exactly as possible to the clinical and pathological facts. But we must remind the reader that we, like *Wagner*, cannot prove all our observations with anatomical verifications according to *Cohnheim's* scheme.

In most cases (our personal observations show this also) the *genuine* amyloid kidney, especially when there is no fever or marked engorgement, emits rather more than the average quantity of urine; perhaps this increase is in keeping with a less active resorption on the part of the medullary substance (*Ribbert*, *Rosenstein*). The specific gravity of the urine is a little diminished, and the colour is pale. As the urine is clear there are naturally fewer sediments. But, on the contrary, the albuminuria is notably (one per cent. and more) much too intense to be only due to circulatory troubles, such as, for example, an elevation of blood-pressure in the kidneys. *Rosenstein* rightly insists on this fact, although he is contradicted by *Dickinson* and *Ewald*. It seems, on the contrary, much more logical to admit that the glomeruli, and with them the interstitial vessels (*Senator*), have acquired by amyloid degeneration the faculty of letting albumin pass by intercepting the passage of the formed elements (*Cohnheim*).

Nevertheless, there is no lack of cases in which the urine has remained for long, even during the whole illness, without albumin, and otherwise normal. Observations of this kind have also been made by *Pleischl* and *Klob*, *Grainger Stewart*, *Rosenstein*, *Litten*, *Naunyn*, *Cohnheim*, *Wagner*, *Fräntzel*, *Straus*, *P. Guttmann*. We observed such frequent examples that we do not understand how they can be considered as rare exceptions. One would be tempted to agree with *Lecorché* that genuine amyloid degeneration, unaccompanied by any other nephritic lesion, does not produce albuminuria; but *Traube* has shown that the same fact of amyloid degeneration of the renal vessels accompanies this symptom. Some of the cases which we have cited can be explained by the fact that amyloid degeneration had just spared the glomeruli (*Cohnheim*, *Straus*, *Rosenstein*, who describes a case), or by the fact that the glomeruli were so degenerated that they took no part in the renal excretion ; but there remains all the same a good number of cases without satisfactory explanation.

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* *Grainger Stewart* first drew attention to the fact that patients suffering from waxy degeneration of the kidney have in addition to the peculiar pallor of chronic kidney disease, a muddy complexion especially noticeable by the brownish rings round the eyelids. He thinks it due to the deposition of pigment in the tissue-element of the skin.—W. H. G.

The urine of *amyloid nephritis* resembles that of chronic diffused nephritis with which the large white kidney is often confounded. The absence of hypertrophy of the heart favours *oliguria*, concentration and cloudiness of the urine ; nevertheless, the symptoms are very variable. Albuminuria is especially plentiful, because it is produced by a double cause, by nephritic lesions and amyloid degeneration. As much as five per cent. has been seen ; we have remarked as maximum three per cent.

In *lardaceous contracted* kidney cardiac hypertrophy sometimes shows itself, sometimes not ; if the former is the case the urine resembles that which we have analysed in the part on "Contracted Kidney," while when the second is the case, polyuria, this important factor, is missing. We will not further discuss the various theories on the subject of albumin, the quantity of urine and the specific gravity.

With regard to the sediments, we know that fatty kidneys seldom produce *hyaline* casts ; when they are accompanied by nephritis one finds casts more or less fatty degenerated ; sometimes (but this is rare) these casts are large and sprinkled with dark granulations ; finally, they are seldom of a genuine epithelial character. We are certain that *Rosenstein's* theory, that all kinds of amyloid nephritis are characterised by a small quantity of sediment, is wrong. In cases of excessive amyloid we have constantly observed a certain number of deposits of all kinds of casts, epithelial casts, fat corpuscles, leucocytes, etc. According to *Wagner*, the waxy casts are found less seldom in the urine than in the kidneys post mortem. Some authors, amongst whom we mention *Grainger Stewart*, *Bartels*, *Wagner*, *Kyber*, have been able to observe amyloid reaction on the epithelial cells and on their attributes, but facts of this kind are, unfortunately, exceptional. We have remarked it ourselves several times, but we cannot warn too strongly against the confusion which is sometimes made with hyaline epithelial cells of the urinary organs, which are, as one knows, sensible to iodine (*cf.* the chapter on gonorrhœa) ; these hyaline casts are physiological. In half the cases one meets with *white blood corpuscles* and in a quarter with *red* (*Wagner*), but the latter are never in a sufficiently large number to render the urine hæmorrhagic.

Senator declares that in amyloid degeneration of the kidney, the urine contains, apart from the serum albumin, a much larger quantity of paraglobulin than the albuminous urine of other forms of nephritis, but *Petri*, *Heynsius* and *Fuhri-Snetlage* have not been able to verify *Senator's* assertion.

The state of urea, uric acid and other uric salts depends less on the state of the kidney than on the changes produced by oxidation (*Bartels*); they have no clinical interest.

According to statistics made by various authors (*Fehr*, *Rosenstein*, *Wagner*), *dropsy*, and especially œdema of the lower extremities and ascites, accompany the fatty kidney in more than half the number of cases. Œdema of the feet, together with albuminuria, in persons suffering from phthisis, is justly said to be a sign of lardaceous kidney. It is most difficult, often in fact impossible, to determine what share derives its origin from the principal malady, from the cachexia and the complicating thrombosis of the crural veins, and what share proceeds from renal degeneration. Circulatory disturbances of the vena portæ produced by compression of amyloid lymphatic glands, or by amyloid degeneration of the hepatic vessels, have been declared to be the cause of ascites (*Roberts*, *Murchison*). We, like *Bartels*, cannot agree with *Lecorché* that the pleura and the pericardium are always spared.

Every one agrees as to the scarcity of *hypertrophy of the heart* as a symptom of amyloid kidney. Out of 265 cases *Wagner* only observed it ten times. This rarity concurs with *Cohnheim's* theory as to the mechanism which produces hypertrophy in the affections of the kidney; and in fact all the conditions indispensable to an elevation of the blood-pressure are wanting in persons suffering from amyloid kidney, who are always more or less cachectic; they are more often seen in syphilitic patients, who feel themselves well, and who suffer from granular fatty kidney, or in those just at the onset of the disease (*Friedreich*, *Böckmann*); they are also found in consumptive patients, whose pulmonary processes recede with the apparition of amyloid degeneration, and whose nutrition is ameliorated by the development of the amyloid kidney (*Ebstein*).

In general cerebral hæmorrhages, retinal lesions and uræmia are as rare as cardiac hypertrophy. *Rosenstein* has only twice observed uræmic accidents and we have only remarked them once. This is explained by the fact that the quantity of urea excreted in the urine differs very little from the average (*Bartels*, *Fleischer*).

The patients are much more often subject to violent *vomitings;* but it is difficult to say if the latter are of a uræmic nature, if they are due to an amyloid degeneration of the digestive tract or to circulatory troubles of the portal system (*Whitaker*). It is the same with diarrhœa, which resists all rules and treatment. Enteritis, evidently, also accompanies these functional disturbances.

The secondary intestinal lesions, of which we have already spoken, are much rarer in the waxy kidney than in diffused nephritis or in contracted kidney.

The *commencement* of the illness is, with rare exceptions, slow and insidious (*Bull* has seen it appear some days after the primary affection). According to *Bartels'* observations, with which we agree, the malady commences with slight intermittent albuminuria. The duration depends on the causal malady; in consumptive patients it usually is of short duration, and generally ends fatally in the course of a year. But it is otherwise with primary amyloid degeneration, syphilis, diseases of the bones, malaria, especially in the upper classes, where all necessary luxuries can be procured. One has seen the illness last ten years and more with rich persons. *Gerhardt* gives a more favourable prognosis for children. We have seen consumptives, whose death has been expected for weeks and who became amyloid, gain strength and live for months. Nevertheless, considerable loss of albumin and derangements of the digestive organs do not occur without shattering the health of the patient, and it is to these facts that one should partly attribute anæmia and marasmus. Death is caused by the primary disease, by extreme cachexia, sometimes by violent diarrhœa, rarely by uræmia.

Diagnosis.—Except in rare cases in which casts and sediments present amyloid reaction, a certain diagnosis, based on an examination alone of the urine, is never possible; it is preferable to have recourse to etiology. And even in this latter case, if one finds oneself in the presence of albuminous and dropsical individuals, who, in consequence of long-continued suppuration, have fallen victims to cachexia, one must not be in a hurry to affirm an amyloid kidney at the commencement, for we have seen that these conditions can easily cause chronic diffused nephritis. When the diagnosis is suspended, and one remarks in cases similar to those which we have just described a *tumour* of the *spleen* or *liver*, this symptom acquires an especial value. All the same, these symptoms do not always suffice to prevent an error in the diagnosis; for one has seen the liver and the spleen hypertrophied during life without amyloid degeneration at the post-mortem. *Rosenstein* warns against drawing decided conclusions from the size of the enlarged organs and the degree of degeneration. It is not known up to what point hyalinosis can participate in hypertrophy of the organ.

When all these accompanying symptoms are missing, the diagnosis can only be based on probabilities. It is evident that it is equally as impossible to diagnose a case of this kind when the urine is normal, as it is to recognise clinically the large white kidney and granular atrophy when the amyloid is only slightly developed; these forms do not, as we have already said, belong to the group of lardaceous kidneys. When the urinary passages are the seat of a catarrhal inflammation, the diagnosis is singularly difficult. Nevertheless, when amyloid urine suffers from sudden changes without apparent cause, the variations can aid the diagnosis.

In order to distinguish amyloid kidney from ordinary contracted kidney one must pay exact attention to the hypertrophy of the heart and to the characteristic pulse, not forgetting that these symptoms can accompany the fatty amyloid kidney. A very light urine, containing little albumin, speaks in favour of genuine granular atrophy.

One cannot warn too much against over-forcing the differential diagnosis regarding pure chronic diffused nephritis, and against trying to separate too finely the three principal forms of lardaceous kidney. *Wagner* feared a confusion between amyloid kidney and renal tuberculosis; it suffices to look for bacilli in the urine. It must be remembered once for all that the scheme which we have just drawn up is still very far from sufficient for a certain diagnosis, and in the majority of cases a serious cliniater would be very reserved in his diagnosis.

Prognosis.—Amyloid kidney usually ends fatally. There are only a few isolated cases where cure is *probable* (*Stewart*, *Bartels*, *Bäumler*, *Gerhardt*, *Eichhorst*, *Wagner*). In spite of the large number of cases which we have had occasion to observe we do not know of one positive cure. *Rosenstein* also doubts the possibility of a cure, even when the process is only commencing. Nevertheless, according to *Litten*, it is possible, by increasing the quantity of the blood and improving the nutrition, to arrest the amyloid process and to bring the kidney back to the first stage of the illness, that is to say, to the hyaline period. This author, for the object of studying this retrocession, placed some pieces of amyloid kidney in the abdominal cavity of animals submitted to his experiments. After some months these little particles became porous, surrounded by abundant connective tissue with extensive vascularisation, and containing numerous giant-cells, which had also penetrated into the amyloid glomeruli and which were doubtless produced by the emigration of leucocytes. The amyloid degeneration was transformed into hyaline degeneration. *Rählmann* also describes a retrogression of an amyloid degeneration of the conjunctiva. The prognosis is more favourable when one has to do with a curable syphilis or surgical affections in children; but we are far from being as sanguine as *Gerhardt*, who considers these cases "as almost certain" to be curable. The duration of the illness depends naturally on the basis of its development. The proportion of excreted albumin can hardly be considered as a measure for progress, suspension and retrogression. The nutritive condition aids the physician much more. A certain number of patients can pursue their ordinary occupations for a considerable length of time. We have seen a syphilitic patient, suffering also from granular fatty kidney, fulfil his duties as custodian.

Treatment.—The best means of combating this affection consists in a rational *prophylaxis*, in surgical treatment of the affections which could have caused it, in an energetic struggle against syphilis or malaria when these affections have caused the malady.

But when the amylosis is firmly established the chances of cure are less. Even in this case one must attack the original affection. Amylosis in consumptive patients is not easy to combat; if the patient suffers from dropsy, dyspepsia, uræmia, one uses the treatment already described. Dropsy seems to be even more impervious to diuresis and diaphoresis than with non-amyloid nephritis. We have seen remarkable, if not lasting results, obtained by frequent scarifications. It seems to us that cutaneous wounds in patients suffering from amyloid cachexia are much less predisposed to suppuration than in persons suffering from *Bright's* disease in general. We do not speak of the treatment of the affection itself. We have no confidence in the pretended specific remedies when the malady is declared; and we consider it useless to enumerate all the remedies more or less sanctioned by tradition.

We have already often mentioned the other two degenerations of the kidney, the parenchymatous disturbance ("cloudy swelling") and fatty degeneration of the epithelial cells, which one must not confuse with fatty infiltration produced by obesity or lipomatosis. Manuals on pathological anatomy and pathology in general treat on these forms, which are besides of no clinical importance if not complicated with ischæmic kidney or nephritis. We content ourselves with reminding that the liver, the kidneys and the heart are the seat of predilection of these two metamorphoses. *Virchow* considered this cloudy swelling (granular degeneration) as an anterior stage of fatty degeneration; but the albuminous reaction of the granulations speaks against this opinion. These granulations, soluble in acetic acid, insoluble in ether, fill the epithelial cells, which are already hypertrophied and degenerated and give to the organ this dull, opaque aspect, which gives it a boiled appearance. Fatty degenerations, which have not been preceded by any parenchymatous swelling, are also to be found; this cellular alteration is not generally followed by adiposity.

The *causes* of these two metamorphoses are identical. They are general and local (infectious diseases, intoxications, alterations of the arterial current, *Zielonko*, *v. Platen*). We will simply add, that when one submits the organ to heat one observes a fatty degeneration, but cloudy swelling does not occur (*Litten*); one sometimes meets with the latter post mortem; it proceeds more often from general infection than from fever. Among the poisons we will only mention phosphorus, arsenic and certain mineral acids. As a type of these intoxications one could take the fatty kidney of individuals poisoned by phosphorus. Finally, one must not forget pernicious anæmia as origin; cachectic maladies (phthisis, cancer) cause rather the disappearance of the fat, with which they over-charge the blood, by producing a fatty infiltration. According to *Rosenstein*, senility without arterio-sclerosis leads also to fatty degeneration and thence to atrophy. The essential cause exists in the diminution of uric acid (*A. Frankel*).

For *coagulative necrosis, cf.* " Hæmorrhagic Infarctus ".

Let us now examine the value of *Senator* and *Lépine's* opinion, who have so strongly proclaimed the immediate relationship between *albuminuria* and degeneration of the renal epithelium. *Senator* has undertaken a series of experiments on this subject and tried to establish a parallel between fatty degeneration produced by phosphorus and albuminuria. If it is the function of epithelial cells of the tubuli to prevent albumin from passing out of the interstitial vessels into the tubuli, one conceives that degeneration of the protoplasma of these cells breaks the barrier which was opposed to albumin, and thus albuminuria is produced. Having seen intense albuminuria appear in diphtheritic nephritis when the kidneys presented no glomerular alteration, but a degeneration of the tubuli of the cortex, we had already recourse to this hypothesis to explain the albuminuria. Nevertheless, seductive as *Senator's* theory may be, one must not forget that *Rosenstein*, as well as ourselves, has observed a good number of total fatty degenerations produced by pernicious anæmia, which were not accompanied by the least trace of albumin; facts of this kind show that degeneration of epithelium is not the only cause of albuminuria.

No fresh light has been thrown on the clinical signification of the metamorphoses of the renal epithelium of toxic origin, and which have been the object of repeated studies, such as *glandular epithelial necrosis, hyaline swelling* (glycogenic degeneration) in diabetes mellitus (*Armanni*, *Ebstein*, *Ehrlich*, *Frerichs*, *J. Straus*). Opinions are still very much at variance as to the relationship existing between this degeneration and the diabetic coma. *Neelsen* and *Fichtner* have recently seen in persons suffering from diabetes, fatty degeneration of the cortex at the same time as glycogenic degeneration of *Henle's* ansae, but without cellular necrosis or interstitial affections. The limits of this book forbid our entering into more histological details on this subject.

II. AFFECTIONS OF THE KIDNEYS LEADING TO SUPPURATION.

Nephritis Suppurativa, Apostematosa, Nephritis Vera, Nephritis Interstitialis, Nephritis Circumscripta, Nephritis with Purulent Centres, Renal Abscess.

These affections were not unknown to *Hippocrates*, and before *Rayer's* day were also included in the group of purulent affections of the pelvis renalis, pyonephrosis and all other sequels of pyelonephritis. The pathogenesis of these forms of suppurative nephritis has been explained in *Virchow* and *Cohnheim's* well-known works on embolic abscesses.

Nephritis ending in suppuration is infinitely rarer than all those which we have studied up to this time; and as surgical intervention is nearly always necessary, this nephritis is more often treated by surgeons; hence the name *surgical kidney.* We have only observed these suppurations in about one-tenth of the cases of diffused nephritis. In the great majority of the cases one has to do with secondary processes.

Etiology and pathogenesis.—Purulence in the kidneys seems to be produced by the blood. That is the case in suppurative nephritis. The origin is:—

(1) *Pyæmic* and *septic* processes in the widest sense of the word. The infection usually spreads in the form of malignant *ulcerative endocarditis.* This endocarditis serves as intermediary between nephritis and the original malady (puerperal fever, septicæmia, acute infectious maladies, actinomycosis (*Israël*), phthisis, etc.). Embolisms of colonies of bacilli, which have already been described by *Bekmann* and demonstrated by *Recklinghausen*, *Klebs*, *Eberth*, *Weigert*, exercise their specific action by developing in general "metastatic" miliary abscesses. Intermediary forms between these kinds of nephritis and those which we have studied under the name of diffused nephritis of septic origin are evident. But these pyæmic processes can produce in the kidney vast purulent centres, veritable abscesses, such as are in all the organs of the body, notably in the lungs and in the liver. *Steven* classified these abscesses into abscesses of small symmetric volume, multiple, situated especially in the cortex, and into abscesses of great volume, constituting a purulent unilateral centre according to their origin (pyæmic, or derived from ulcerous endocarditis); but this classification does not seem to us to agree always with the facts, for here, as elsewhere, we find an infinite variety of intermediary forms which prevent the establishment of a distinct limit. *Böttcher* has shown that a renal abscess was produced through a pulmonary purulent centre by the presence in the kidney of pulmonary elastic fibres.

(2) The pyogenic elements can reach the kidney by the *urinary organs.* With rare exceptions, in which infection reaches the kidneys direct without attacking the pelvis, the latter is generally affected before the kidneys; they form to some extent an intermediary centre between the infection of the urinary organs and that of the renal parenchyma. In other words, the etiology on these suppurations is in pyelitis or pyelonephritis, an occurrence of the greatest practical importance. In order to avoid fastidious repetition, we refer our readers to the chapter treating on these two affections. In general, purulent

nephritis is of a calculous origin, or it is due to some obstacle seated in the ureter opposing the course of the urine (stricture, prostatic enlargement). It can also proceed from spinal complaint; this does not act direct, as *Rayer* declares, but with the intermediary of a secondary cystitis.

(3) Suppurative nephritis is seldom produced by *propagation* of *adjacent* purulent deposits, in consequence of perinephritic phlegmon of the liver, of the spleen, abscesses of the psoas, and of caries of the vertebræ.

(4) Suppuration of the kidney of a *traumatic* origin is also rare. Wounds of this kind are produced by a fall or a blow in the lumbar region with contusion or rupture of the kidney. Death generally occurs from hæmorrhage, or from other visceral lesions, before nephritis has had time to establish itself. *Manoury*, confirming what *Chambers* an age ago observed, rightly remarks that suppuration of wounds of the kidneys is much more rare than is generally supposed, and that, in authentic cases of this kind, it is often produced by medical manipulations (use of the catheter). *Quincke* and *Singer* state that abscesses of the kidney develop themselves exceedingly slowly, and remain latent for years, only manifesting themselves by means of an intercurrent malady, such as typhus, lithiasis, etc.

(5) *Renal tuberculosis* can cause suppurative nephritis.

Finally, there remain a number of cases belonging to the series of suppurative nephritis called idiopathic, the cause of which we cannot yet definitely fix. To this category we must reckon the abscesses occurring in persons suffering from *diabetes*, and where it is not likely that it comes from taking cold. *Rosenstein* cites one of these so-called spontaneous cases in which it was impossible to discover the micro-organisms (*cf.* "Pyelitis").

It has been said that certain very efficacious drugs, such as cantharides, have produced abscesses, but this opinion can be considered as erroneous.

Pathological anatomy.—When metastatic nephritis is of a pyæmic or septic origin, or is due to ulcerous endocarditis, one meets, especially in both kidneys, with *miliary* embolic deposits. Where the deposits are situated on the surface of the organ, the capsule is more adherent. The dull surface and section of the hypertrophied organs is studded with small grey and red spots, which sometimes attain the size of a grain of mustard seed; these purulent centres are of a greyish-yellow colour, and, according to our personal observation, nearly always surrounded by a hyperæmic or even hæmorrhagic zone. In the cortex these centres are round or wedge formed; in the medullary substance they are linear. When these centres extend one could consider the organ covered with genuine variola. Even depressions are here and there to be found. Under the microscope these grains appear to result from cellular infiltrations; their centres contain smaller or larger colonies of *micrococci* (streptococcus, staphylococcus). Their seat of predilection is round the malpighian corpuscles and the veins. *Gram's* method of various colorations gives very good preparations. One then perceives the microorganisms in the glomerular loops, in the capillaries surrounding the arteries and veins, and, finally, in the interstices of the cortex and of the medullary substance. One has at the same time observed infarcts obstructing the arteries. The epithelial cells occupying the pericapsular and perivascular regions, surrounded by purulent deposits, invariably form necrosis and increase the abscess. When genuine renal, so-called idiopathic, *abscesses* produced by a pyæmic process, as those which produce hepatic and pulmonary suppurations, exist, one finds on section more or less extended regions where the tissues have completely disappeared, and are replaced by large purulent centres, the walls of which, at first wrinkled, become quite smooth. The intact parts of the kidney are generally very hyperæmic, the cortex becomes confluent with the medullary substance, without one's being able to establish a boundary zone between them, and one meets with more or less marked development of diffused nephritis. By progressive suppuration on the periphery and confluxion of the enlarged abscesses, vast destructions of the organ attend; one has even observed the kidney to be transformed into a sack of pus. *Ewald* cites a case of this kind in which suppuration had occupied the right half of the abdomen, and even encroached on to the other half. These "pyonephrosis" are generally the consequence of pyelonephritis. When communication with the pelvis exists, or when the latter is likewise attacked, it is often very difficult, even sometimes impossible, to say whether the departure is to be sought for in suppuration of the kidney or in pyelitis with inflammation spread to the renal parenchyma. Finally, when the suppuration of the kidney extends further, they make way for perinephritic phlegmons, retro-peritoneal, purulent abscesses, frequently followed by fistules in the lumbar or anterior abdominal region; sometimes the suppuration even perforates into the adjacent organs, and extends into the liver, the colon, the small intestines, the bronchi, and, finally, into the peritoneum. On recovery the abscesses empty themselves, and the suppuration is replaced by granulations and formation of callosity. In a number of cases the abscess becomes circumscript, and the pus is thickened into caseous and chalky material. Amyloid degeneration frequently concurs. Contrary to the opinions of *Rosenstein* and *Ebstein*, we have observed bilateral amyloid degeneration even when suppurative nephritis has only attacked one side.

For further details on the subject of *paranephritic phlegmons*, which, as we have seen, can be the origin of suppurative nephritis, we refer our readers to the chapter on this subject, and for details on pathological anatomy of suppurative nephritis developed by the intermediary of the urinary organs, to the part on pyelitis and pyelonephritis, and also on nephrolithiasis. In most cases the suppurations are unilateral (see "Renal Tuberculosis").

Finally, we leave to the treatises on surgery to describe the pathological anatomy of the *traumatic lesions* of the kidney, which are usually unilateral; we content ourselves with saying that, when death occurs at the commencement, large extravasations of blood are found in the ureter, in the bladder and in the kidney, the tissues of which are generally more or less ragged and disintegrated. When nephritis has had time to develop itself, the organ is swollen and hyperæmic and is saturated by a brown-coloured liquid; the parenchyma contains numerous hæmorrhagic deposits, in the midst of which purulent centres appear; the latter occupying especially the cortex. These deposits have a very decided tendency to confluence and extension. In very serious cases, especially when they are accompanied by infiltration of urine, the entire organ mortifies and is transformed into a kind of pulpous and purulent mass.

Symptomatology and diagnosis.—The *miliary* metastatic abscesses are not easy to diagnose during life, because their evolution modifies in no way the course of the original disease. When the physician has been happy enough to diagnose infectious endocarditis he must suppose the abscesses to be on the side of the kidney, and his diagnosis is often confirmed; in other cases miliary abscesses are found unexpectedly at the post-mortem.

Even large renal abscesses, the result of pyæmic illnesses, are difficult to diagnose as long as they remain confined in the renal parenchyma, because they do not exercise any characteristic influence on the course of pyæmia.

The other forms of suppurative nephritis, as far as they are of no traumatic origin, even often pass unnoticed by the expert physician, especially when they are consecutive to affections of the urinary passages and of renal lithiasis. The symptoms of the causal affection often mask the renal processes themselves. In fact "genuine" cases of suppurative nephritis are exceptional, and *Rosenstein* was right when he said some twenty years ago, "in consequence of the scarcity of pure cases, description must remain incomplete". More marked than the symptoms are the *complications* of the illness.

The abscesses in question rarely take an acute course. A considerable elevation of temperature, rigor, vomiting, pains in the kidneys, dyspepsia, are the initial symptoms. Nevertheless, it occurs that renal abscesses commence brusquely with these symptoms and then take a chronic course, so much so that there is no difference between them and the chronic ones from the commencement; these latter are almost invariably consecutive. Sometimes the fever ceases, or decreases, and is only remarkable by slight attacks of shivering. We have frequently noticed great oscillations in the temperature, variations from 36° to 39° C., in cases of serious septicæmia from 40° to 41° C. In cases of ulcerative endocarditis we have observed at various intervals sudden changes of 5° to 6° C. within a few hours. In half of the cases the patients do not complain of pains in the kidney. Nevertheless, we have seen patients tortured day and night for weeks and months at a time. But it is not thus that these matters generally end; usually the pains are lessened by repose, and increase with the least movement. They often radiate to the loins, the inguinal regions, to the testicles, to the labia pudendi and to the femur of the same side. Reflex pains are often taken for real renal pains. We have seen a woman scream during palpation of a sound kidney and bear palpation of a diseased kidney without much pain. Examination, especially when made by a rough or inexperienced physician, is excessively painful; we have seen patients who have been chloroformed scream for several hours after they awoke. Sometimes, to soothe their pain, they keep the femur of the affected side in permanent flexion, thus preventing the compression of the nerves. This nephritis is distinguished from coxalgia by the absence of pain in passive flexion, and the contrary by extension of the femur (*Roberts*). We warn against too much reliance on this differential diagnosis.

In most cases the character of the *urine* is tolerably complicated. Firstly, it must be remembered that diffused nephritis can provoke pyrexial albuminuria; but suppurations are most frequently accompanied by complicated catarrhs of the urinary organs. In pure suppurative nephritis there is no albuminuria nor numerous formed elements (casts, renal epithelium). One has sometimes observed anuria for days; we ourselves have never remarked this, and it seems to us that anuria is no clinical symptom of uncomplicated suppurative nephritis, the kidneys having always certain regions of the parenchyma capable of fulfilling their functions. We rather believe that anuria accompanies certain complications produced by nephritis, such as lesions of the urinary organs, destruction of one kidney by other causes producing an intense suppuration of the other one. But, on the other hand, nothing is more frequent than oliguria and strangury, even when the urinary organs are intact. For details on pyuria and hæmaturia in consequence of a complicated pyelitis we refer our readers to the chapter on this subject.

When urine deposits all of a sudden abundant *purulent sediments*, this purulence generally indicates a rupture of the abscess into the pelvis renalis. The masses of pus are usually tinted with blood, they are sometimes even ammoniacal and fœtid ; they disappear rapidly. Nevertheless, in a certain number of cases one observes symptoms of a chronic cysto-pyelitis. The greater number of cases of suppurative nephritis are only diagnosed at the moment where purulent discharges take place. But one must not forget that these latter can be caused by a paranephritic phlegmon or an abscessus congestivus opening into the urinary passages.

The presence of fragments of parenchyma (*Taylor*, *Wiederhold*), veritable *renal sequestra*, in which the microscope shows glomeruli and tubuli uriniferi, proves the renal origin of the pus ; unfortunately for the cliniater these facts are exceptional. It is less uncommon to find fragments of necrotic pyramids (*Przewóski*).

A third objective symptom appears, especially when the malady is of old date, and the suppuration, having surrounded a large part of the kidney, has met with an obstacle. In fact when one explores the lumbar region by means of palpation one feels a *tumour* which is seldom so fluctuating that one can affirm its contents ; we shall see later on (see "Hydronephrosis") which are the differential characters of this tumour. Sometimes when fluctuation is wanting a trial puncture produces pus ; therefore one must not confound a renal abscess with extra-renal phlegmons, especially with lesions of the peritoneum (see "Hydronephrosis").

In some cases one observes *motor* and sensitive derangements of the corresponding, sometimes even of both, lower extremities. But the number of these forms of *paralysis* and urinary paraplegia is much too small for one to be decided as to the genesis of these accidents, which are, besides, strangers to diffused nephritis. One presumes a compression of the corresponding nerve trunks (*Simon*), or a reflex paralysis (*Stanley*, *Leroy*, *Brown-Séquard*, *Levinson*) ; and in basing on the physiological and clinical experiences one has invoked the considerable influence which renal abscesses exercise on the organism. Certain anatomical facts, such as profound lesions of the sciatic nerve and of the spinal cord (*Leyden*, *Kussmaul*), seem to confirm this latter hypothesis. *Rosenstein* attributes in a great measure this paraplegia to the radiation of the suppurative process along the nerves, *i.e.*, to neuritis ascendens ; in all the cases urinary organs were attacked, according to him, *Bright's* disease never leads to these lesions. We are not of *Rosenstein's* opinion.

We have already spoken of the *opening* of the renal abscess into the pelvis, a favourable and frequent occurrence. Numerous are the tracts which the pus forms for itself (see "Pathological Anatomy"). When it makes its issue at the back and at the outside of the kidney, the lumbar region presents a red and fluctuating tumour ; when it flows in great quantities, the urine is ammoniacal and becomes decomposed. The prognosis is much more serious when the pus perforates the retro-peritoneal connective tissue and forms vast phlegmons and congested abscesses which point to the inguinal or perineal regions. We have seen pus flow into the intestines, the liver and the lungs. In the first case one arrives at a diagnosis by the mixture of fæcal matters with pus, and in the latter the patients are attacked suddenly with dyspnœa and expectorate pus with a urinous odour on coughing. Such cases are not always fatal, but, when perforation into the abdominal cavity takes place, fatal peritonitis rapidly occurs.

The happiest termination is that in which the purulent deposit empties itself outwardly or into the urinary passages and gives place to granulations and to cicatricial tissues. Nevertheless, the purulent effusion does not always dry, it spreads into other organs, which give place to numerous fistulæ, and after many months the patient succumbs to a hectic fever, to extreme cachexia, amyloid disease, or to phthisis complicating nephritis.

When the disease precedes or follows lesions of the urinary organs it takes, especially towards the end, a *typhous* character; in these cases the patient suffers from nervous depression, with sudden intercurrent exasperations, delirium, oliguria, rigor, dyspepsia, diarrhœa, dryness in the mouth; the patient is exhausted, low-spirited, the pulse is filiform; little by little the patient is bathed in viscous perspiration, falls into coma and death takes place. It is still difficult for us to say up to what point "urosepsis" is connected with uræmia. When the urine undergoes an ammoniacal fermentation, and one does not observe cramp, dropsy, nor alteration in the features, and when the patients are attacked by violent rigor, uræmia does not usually exist (*Treitz, v. Jaksch, Sée*); for here it is the alkaloid substances resulting from the decomposition of urine in the urinary tracts, and not in the kidneys, which are resorbed. It has been tried to attribute these symptoms to ammoniæmia, but they did not at all agree with those which one observes in the experimental ammoniacal intoxication, which always accompanies convulsions (*Rosenstein*). We cannot insist enough on the fact that in the three last years we have noticed these symptoms, even when ammoniacal decomposition is missing; further, we have seen convulsions interrupt from time to time the comatous state, and they seemed to coincide precisely with a marked diminution of urine (it is true that the latter is not always to be ascertained in these cases). Thus we have observed cases absolutely analogous to those which we have described in chronic uræmia, which accompanies diffused nephritis and the atrophied kidney. According to *Bouchard's* works on the various urinary poisons as agents of auto-intoxication, these symptoms could well be of a uræmic nature, all the more as *Senator* and *Saundby* have latterly declared that they have seen symptoms absolutely identical with those mentioned by *Kussmaul* in the diabetic coma. With these exceptions we agree with *Rosenstein* and *Jaksch* that the resorption of the putrefied urine or decomposed matter must be placed in the foreground. One has seen patients who have completely lost one kidney, and where the other was in full suppuration, remain several days anuric without cerebral symptoms showing themselves (*Wilmot*).

For details on the symptomatology and the particular course of renal traumata and *traumatic* nephritis see treatises on surgery. In general, traumatic lesions are immediately followed by hæmaturia, sometimes even the bladder contains large clots of coagulated blood, and it is only some days after the accident that pyuria declares itself, which is the characteristic symptom of renal suppuration; still pyuria is often missing. Even in traumatic nephritis one frequently observes an insidious course during months at a time (*Singer*), in which the urine is constantly hæmorrhagic and purulent. In exceptional cases one has been able, by catheterising the ureters, to define the source of the hæmorrhage.

For details on diagnosis we refer our readers to the chapters on "*Pyelo-nephritis*" and "*Paranephritis*".

Prognosis.—The prognosis is naturally very serious when the disease is of a pyæmic or septicæmic origin. As one cannot foresee the issue of the malady the prognosis must always be held in reserve. We have seen in the preceding part which were the happiest terminations; we will, therefore, not return to this. We have never seen cases cured in a few days. Trauma, although not dangerous, gives way to an insidious form of nephritis, which can last for months and even for a year. When the patient is attacked by hectic fever, and cachexia appears at the same time as the symptoms of urosepsis just described, the prognosis is singularly aggravated, even when anti-uræmic medication has somewhat improved the general condition, and diarrhœa (*Assmuth*) has been already allayed for some time.

Treatment.—Treatment is always powerless when the renal abscesses are of a pyæmic and septicæmic origin. When suppurative nephritis results from propagation, by means of the urinary organs, of the inflammatory processes of pyelitis, cystitis, or stricture of the urethra, or even when it is in consequence of a paranephritic phlegmon, one sometimes attains a good result by attacking the primary cause.

In general, acute forms demand repose and a rational antifebrile treatment, in which the obligatory applications of ice are naturally included, which, however, have no particular result. Local bleedings have often an excellent effect, but we only recommend them at the onset of the malady and the patient being of a robust constitution. When the malady takes a chronic course, and the patient presents hectic symptoms, strengthening nourishment must be prescribed as well as baths; if the pain is very violent one uses cataplasms, morphia, opium (also in suppositories), chloral; finally, narcotics in general, which must be sometimes administered for some time and in large doses. We have explained the value of anti-febrile treatment in the part on "Acute Nephritis". The drug which seems to us to be the most reliable is quinine, but it is comprehensible that the same does by no means arrest the process of suppuration.

If an abscess forms itself or a purulent sac, surgical intervention is necessary, although *Minoury* warns against operating too soon, especially when the disease is of traumatic origin (*cf.* the part on "Calculous Kidney").

End of Vol. I.

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